DOCUMENT RESUME

ED 070 647 SE 015 367

TITLE	Air Quality Criteria for Particulate Matter.
INSTITUTION	National Air Pollution Control Administration (DHEW), Washington, D.C.
REPORT NO	NAPCA-AP-49
PUB DATE	Jan 69
NOTE	218p.
EDRS PRICE	MF-$0.65 HC-$9.87
DESCRIPTORS	*Air Pollution Control; *Environmental Criteria; Environmental Influences; *Literature Reviews; *Matter; Pollution; Quality Control; Standards

ABSTRACT

To assist states in developing air quality standards, this book offers a review of literature related to atmospheric particulates and the development of criteria for air quality. It not only summarizes the current scientific knowledge of particulate air pollution, but points up the major deficiencies in that knowledge and the need for further research. Focused upon is total particulate matter of the kind normally measured by high-volume and paper-tape sampling methods and by dustfall collection. Further, it considers the effects of particulate matter in conjunction with some gaseous materials where important synergistic effects are observed. Methods of measuring the effects of particulate matter on meteorological conditions, atmospheric visibility, materials, and vegetation are documented, as well as the resulting economic loss. Public awareness of air pollution and the role of particulate matter in the odor problem are assessed. There is a chapter on the respiratory system, discussing particulate deposition therein and removal therefrom, necessary to understanding the final chapters which survey toxicological effects of particulate matter and the epidemiological data for man and animals. A glossary of terms, lists of symbols, abbreviations, and conversion factors for various units of measurement, author index, and subject index are provided. (BL)

ED 070647

U.S. DEPARTMENT OF HEALTH
EDUCATION & WELFARE
OFFICE OF EDUCATION
THIS DOCUMENT HAS BEEN REPRODUCED EXACTLY AS RECEIVED FROM THE PERSON OR ORGANIZATION ORIGINATING IT. POINTS OF VIEW OR OPINIONS STATED DO NOT NECESSARILY REPRESENT OFFICIAL OFFICE OF EDUCATION POSITION OR POLICY.

AIR QUALITY CRITERIA
FOR
PARTICULATE MATTER

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service

SE 015 367

ERRATA FOR
AIR QUALITY CRITERIA FOR PARTICULATE MATTER

Page 37, Fig. 2-2: Fill in dots on right-hand curve. (to agree with key)

Page 65, col. 2, para. 2, line 3: "sol serve as . . ." should be "sols serve as . . . "

Page 68, col. 1, line 2: ". . . Kearney,[10] it" should be ". . . Kearney.[10] It"

Page 68, col. 2, Fig. 4-2: The bottom curve in the figure (i.e., State College, Pa.) should contain four filled-in circles; i.e., the first circle should be filled in.

Page 69, col. 1, para. 3, line 2: ". . . where pervious metals" should be ". . . where precious metals"

Page 69, col. 2, para. 3, lines 6-7: "buildings in burning soot-producing fuel cities.[15-17]" should be "buildings in cities burning soot-producing fuel.[15-17]"

Page 69, col. 2, para. 3, 18th line: "less resistant tyes of mason-" should be "less resistant types of mason-"

Page 69, col. 2, para. 4, line 5: "clean the smoke and" should be "clean the soot and"

Page 72, col. 1, para. 1, line 11: "conditions; show . . ." should be "conditions show . . ."

Page 73, col. 1, para. 2, lines 3-4: "are due only to differences in particulate matter, since there are significant . . ." should be "are due not only to differences in particulate matter, but also to significant . . ."

1A

Page 89, col. 2, para. 2, line 11: "Czaja$^{3-6}$" should be "Czaja^{5,8,15,16}"

Page 90, col. 2, line 1: "the rate of 0.47 mg/cm^2-day and then ex-" should be "the rate of 0.47 mg/cm^2-day, a relatively high rate, and then ex-"

Page 90, Fig. 6-1, line 1 of title: "Damp" should be "Dry"

Page 92, col. 2, para. 2, lines 6 and 7: "0.75 g/m^2-day to 1.5 g/m^2-day" should be "750 mg/m^2-day to 1500 mg/m^2-day."

Page 92, col. 2, para. 3, lines 8, 10, 11, and 12: "1.5 g/m^2-day . . . 2/5 g/m^2-day . . . 3.8 g/m^2-day" should be "1500 mg/m^2-day . . . 2500 mg/m^2-day, and 3800 mg/m^2-day."

Page 96, ref. 6 and 22: "Allgem. Forstz" should be "Allgem. Forst Zeitschrifte."

Page 139, col. 2, line 13: "hermatite" should be hematite"

Page 162, col. 1, para. 1, line 3: "Anderson^{30}" should be "Anderson^{32}"

Pages 171-175, Table 11-6, 4th col. heading: "(in brackets) or dustfall tons/mi^2-mo" should be "COH (in brackets) or dustfall, tons/mi^2-mo."

Page 186, col. 2, para. 3, line 12: "tons/m^2-month)" should be "tons/mi^2-mo)"

Page 188, col. 2, para. 5, line 6: "C-4)" should be "C-5)."

Page 188, col. 2, para. 6, line 8: "C-3)" should be "C-5)."

Page 188, col. 2, para. 7, line 2: ". . . particulates on a 24-hour average," should be ". . . particulates (6-month average)."

Page 189, col. 1, line 1: "Section C-5)" should be "Section C-2d)"

Page 189, col. 1: Add to line 8 "(British Data; see Chapter 11, Section C-3)."

Page 189, col. 1, para. 3, line 6: "C-2" should be "C-2b."

Page 189, col. 1, para. 4, line 4: ". . . about 30 mg/cm^2-mo," should be "0.3 mg/cm^2-mo."

AD 070647

AIR QUALITY CRITERIA
FOR
PARTICULATE MATTER

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Consumer Protection and Environmental Health Service
National Air Pollution Control Administration
Washington, D.C.
January 1969

2

National Air Pollution Control Administration Publication No. AP-49

Preface

Air quality criteria tell us what science has thus far been able to measure of the obvious as well as the insidious effects of air pollution on man and his environment. Such criteria provide the most realistic basis that we presently have for determining to what point the levels of pollution must be reduced if we are to protect the public health and welfare.

The criteria that we can issue at the present time do not tell us all that we would like to know. If all of man's previous experience in evaluating environmental hazards provides us with a guide, it is likely that improved knowledge will show that there are identifiable health and welfare hazards associated with air pollution levels that were previously thought to be innocuous. As our scientific knowledge grows, air quality criteria will have to be reviewed and, in all probability, revised. But the Congress has made it clear that we are expected, without delay, to make the most effective use of the knowledge we now have.

The Air Quality Act of 1967 requires that the Secretary of Health, Education, and Welfare ". . . from time to time, but as soon as practicable, develop and issue to the States such criteria of air quality as in his judgment may be requisite for the protection of the public health and welfare. . . . Such criteria shall . . . reflect the latest scientific knowledge useful in indicating the kind and extent of all identifiable effects on health and welfare which may be expected from the presence of an air pollution agent. . . ."

Under the Air Quality Act, the issuance of air quality criteria is a vital step in a program designed to assist the States in taking responsible technological, social, and political action to protect the public from the adverse effects of air pollution.

Briefly, the Act calls for the Secretary of Health, Education, and Welfare to define the broad atmospheric areas of the Nation in which climate, meteorology, and topography, all of which influence the capacity of air to dilute and disperse pollution, are generally homogeneous.

Further, the Act requires the Secretary to define those geographical regions in the country where air pollution is a problem—whether interstate or intrastate. These air quality control regions will be designated on the basis of meteorological, social, and political factors which suggest that a group of communities should be treated as a unit for setting limitations on concentrations of atmospheric pollutants. Concurrently, the Secretary is required to issue air quality criteria for those pollutants he believes may be harmful to health or welfare, and to publish related information on the techniques which can be employed to control the sources of those pollutants.

Once these steps have been taken for any region, and for any pollutant or combination of pollutants, then the State or States responsible for the designated region are on notice to develop ambient air quality standards applicable to the region for the pollutants involved, and to develop plans of action for implementing the standards.

The Department of Health, Education, and Welfare will review, evaluate and approve these standards and plans, and once they are approved, the States will be expected to take action to control pollution sources in the manner outlined in their plans.

At the direction of the Secretary, the National Air Pollution Control Administration has established appropriate programs to carry out the several Federal responsibilities specified in the legislation.

Air Quality Criteria for Particulate Matter is the culmination of intensive and dedicated effort on the part of many persons—so many, in fact, that it is not practical to name all of them.

In accordance with the Air Quality Act, a National Air Quality Criteria Advisory Committee was established, having a membership broadly representative of industry, universities, conservation interests, and all levels of government. The committee, whose members are listed following this discussion, provided invaluable advice on policies and procedures under which to issue criteria, and provide major assistance in drafting this document. To facilitate the committee's work, subcommittees were formed to provide intensive efforts relating to specific pollutants—initially for particulate matter and for sulfur oxides.

With the help of the Subcommittee on Particulate Matter, expert consultants were retained to draft portions of this document, with other segments being drafted by staff members of the National Air Pollution Control Administration. After the initial drafting, there followed a sequence of review and revision by the subcommittee, and by the full committee, as well as by individual reviewers especially selected for their competence and expertise in the many fields of science and technology related to the problems of atmospheric pollution by particulate matter. These efforts, without which this document could not have been completed successfully, are acknowledged individually on the following pages.

As also required by the Air Quality Act of 1967, appropriate Federal departments and agencies, also listed on the following pages, were consulted prior to issuing this criteria document. A Federal consultation committee, comprising members designated by the heads of seventeen departments and agencies, reviewed the document, and met with staff personnel of the National Air Pollution Control Administration to discuss their comments.

This Administration is pleased to acknowledge the efforts of each of the persons specifically named, as well as the many not named who contributed to the publication of this volume. In the last analysis, however, the National Air Pollution Control Administration is responsible for its content.

JOHN T. MIDDLETON,
Commissioner, National Air Pollution
Control Administration

NATIONAL AIR QUALITY CRITERIA ADVISORY COMMITTEE

Chairman
DR. JOHN T. MIDDLETON, *Commissioner*
National Air Pollution Control Administration

Dr. Herman R. Amberg
Manager, Manufacturing Services Dept.
Central Research Division
Crown Zellerbach Corp.
Camas, Wash.

Dr. Nyle C. Brady
Director, Agricultural Experiment Station
Cornell University
Ithaca, N.Y.

Dr. Seymour Calvert
Director, Statewide Air Pollution Research Center
University of California, Riverside
Riverside, Calif.

Dr. Adrian Ramond Chamberlain
Vice President
Colorado State University
Fort Collins, Colo.

*Dr. Raymond F. Dasmann
Senior Ecologist
Conservation Foundation
Washington, D.C.

Mr. James R. Garvey
President and Director
Bituminous Coal Research, Inc.
Monroeville, Pa.

Dr. David M. Gates
Director
Missouri Botanical Gardens
St. Louis, Mo.

Dr. Neil V. Hakala
President
Esso Research & Engineering Co.
Linden, N.J.

Dr. Ian T. Higgins
Professor, School of Public Health
The University of Michigan
Ann Arbor, Mich.

Mr. Donald A. Jensen
Executive Engineer
Ford Motor Co.
Dearborn, Mich.

Dr. Herbert E. Klarman
Professor of Public Health Administration and Political Economy
School of Hygiene and Public Health
Johns Hopkins University
Baltimore, Md.

Dr. Leonard T. Kurland
Professor of Epidemiology
Mayo Graduate School of Medicine
Head, Medical Statistics
Epidemiology and Population Genetics Section, Mayo Clinic
Rochester, Minn.

Dr. Frederick Sargent II
Dean, College of Environmental Sciences
University of Wisconsin
Green Bay, Wis.

Mr. William J. Stanley
Director, Chicago Department of Air Pollution Control
Chicago, Ill.

---

* Resigned, October 14, 1968.

CONTRIBUTORS AND REVIEWERS

Dr. Donald F. Adams
Head, Air Pollution Research Division
College of Engineering
Washington State University
Pullman, Wash.

Dr. Mary O. Amdur
Associate Professor of Toxicology
Department of Physiology
School of Public Health
Harvard University
Boston, Mass.

Dr. Rodney R. Beard
Executive Head, Department
of Preventive Medicine
Stanford University Medical School
Palo Alto, Calif.

Dr. Francis E. Blacet
Emeritus Professor of Chemistry
University of California at Los Angeles
Los Angeles, Calif.

Dr. L. J. Brasser
Head, Atmospheric Pollution Division
Research Institute for
Public Health Engineering
Delft, The Netherlands

Dr. Leslie A. Chambers
Professor and Chairman
Department of Environmental Health
School of Public Health
University of Texas
Houston, Tex.

Dr. Robert Charlson
Research Associate Professor of
Atmospheric Chemistry
Department of Civil Engineering
University of Washington
Seattle, Wash.

Dr. Morton Corn
Associate Professor, Department of
Occupational Health
Graduate School of Public Health
University of Pittsburgh
Pittsburgh, Pa.

Dr. Ellis F. Darley
Plant Pathologist
Statewide Air Pollution Research Center
University of California at Riverside
Riverside, Calif.

Dr. Arthur DuBois
Department of Physiology
Graduate School of Medicine
University of Pennsylvania
Philadelphia, Pennsylvania

Dr. James G. Edinger
Professor of Meteorology
University of California
Los Angeles, Calif.

Dr. Lars Friberg
Chief, Department of Hygiene
Karolinska Institute of Hygiene
Stockholm, Sweden

Dr. Sheldon K. Friedlander
Professor of Chemical Engineering
and Environmental Health Engineering
California Institute of Technology
Pasadena, Calif.

Dr. John R. Goldsmith
Chief, Environmental Hazards
Evaluation Unit
Department of Public Health
State of California
Berkeley, Calif.

Dr. Leonard Greenburg
Professor of Preventive and
Environmental Medicine
Albert Einstein College of Medicine
New York, N.Y.

Dr. Alexander Goetz
Senior Staff Consultant
National Center for Atmospheric
Research
Altadena, Calif.

Dr. Paul Gross
Director, Research Laboratory
Industrial Hygiene Foundation of
America, Inc.
Mellon Institute
Pittsburgh, Pa.

Dr. Harry Heimann
Research Associate, Department of Physiology
School of Public Health
Harvard University
Boston, Mass.

Dr. Walter W. Holland
Professor, Department of Clinical Epidemiology and Social Medicine
St. Thomas' Hospital Medical School
University of London
London, England

Mr. John H. Jacobs
Principal Research Physicist
Bell & Howell Research Center
(Chicago, Ill.)
Pasadena, Calif.

Dr. P. E. Joosting
Medical Service
Research Institute for Public Health Engineering
Delft, The Netherlands

Mr. Elmer R. Kaiser
Senior Research Scientist
School of Engineering and Science
New York University
New York, N. Y.

Dr. Herbert Landesman
Consulting Chemist
Pasadena, Calif.

Dr. Phillip A. Leighton
Emeritus Professor of Chemistry
Stanford University
Palo Alto, Calif.

Dr. Mark H. Lepper
Professor of Preventive Medicine
University of Illinois College of Medicine
Chicago, Ill.

Mr. Robert H. Linnell
Staff Associate, Departmental Science Development Section
National Science Foundation
Washington, D.C.

Mr. Benjamin Linsky
Professor, Department of Civil Engineering
West Virginia University
Morgantown, W. Va.

Dr. James P. Lodge
Program Scientist, National Center for Atmospheric Research
Boulder, Colo.

Dr. Thomas C. Lloyd, Jr.
Associate Professor, Department of Physiology
School of Medicine
Case Western Reserve University
Cleveland, Ohio

Mr. John A. Maga
Executive Officer
Air Resources Board, State of California
Sacramento, Calif.

Dr. Roy McCauldin
Professor, Department of Environmental Engineering
College of Engineering
University of Florida
Gainesville, Fla.

Dr. Herbert C. McKee
Assistant Director, Department of Chemistry and Chemical Engineering
Southwest Research Institute
Houston, Tex.

Dr. Paul E. Morrow
Professor of Radiation Biology and Biophysics
School of Medicine and Dentistry
University of Rochester
Rochester, N. Y.

Dr. Edward D. Palmes
Professor of Environmental Medicine
Institute of Environmental Medicine
New York University Medical Center
New York, N. Y.

Dr. James N. Pitts, Jr.
Professor of Chemistry
University of California at Riverside
Riverside, Calif.

Dr. Walter A. Quebedeaux, Jr.
Director, Harris County Air and Water Pollution Control Division
Houston, Tex.

Dr. Donald D. Reid
Professor of Epidemiology and Director of Department
Department of Medical Statistics and Epidemiology
London School of Hygiene and Tropical Medicine
London, England

Dr. Elmer Robinson
Chairman, Environmental Research Department
Stanford Research Institute
Menlo Park, Calif.

Dr. Stanley N. Rokaw
Chief, Pulmonary Research Section
Rancho Los Amigos Hospital
Los Angeles, Calif.

Dr. August T. Rossano
Professor, Air Resources Program
Department of Civil Engineering
University of Washington
Seattle, Wash.

Mr. Jean J. Schueneman
Chief, Division of Air Quality Control
Maryland State Department of Health
Baltimore, Md.

Dr. Wayne T. Sproull
Consultant in Physics
Pasadena, Calif.

Dr. Gordon H. Strom
Department of Aeronautical Engineering
College of Engineering
New York University
New York, N. Y.

Dr. O. Clifton Taylor
Associate Director
Statewide Air Pollution Research Center
University of California at Riverside
Riverside, Calif.

Dr. Moyer D. Thomas
Editor, Inter-Society Committee Manual of Methods for Ambient Air Sampling and Analysis
Riverside, Calif.

Dr. Amos Turk
Professor, Chemistry Department
The City College of the City University of New York
New York, N. Y.

Mr. Hans K. Ury
Special Consultant
Environmental Hazards Evaluation Unit
California State Department of Public Health
Berkeley, Calif.

Mr. Ralph C. Wands
Director, Advisory Center on Toxicology
National Research Council
Washington, D.C.

Dr. Richard P. Wayne
Oxford University
London, England
Visiting Professor in Photochemistry
University of California at Riverside
Riverside, Calif.

Dr. Phillip W. West
Professor of Chemistry
College of Chemistry and Physics
Louisiana State University
Baton Rouge, La.

Dr. Warren Winkelstein, Jr.
Professor and Head, Division of Epidemiology
School of Public Health
University of California
Berkeley, Calif.

Dr. Harold Wolozin
Professor and Chairman
Economics Department
University of Massachusetts
Boston, Mass.

Mr. John E. Yocom
Senior Research Engineer
Travelers Research Center, Inc.
Hartford, Conn.

FEDERAL AGENCY LIAISON REPRESENTATIVES

Department of Agriculture
Kenneth E. Grant
Associate Administrator
Soil Conservation Service

Department of Commerce
Paul T. O'Day
Staff Assistant to the Secretary

Department of Defense
Colonel Alvin F. Meyer, Jr.
Chairman
Environmental Pollution Control Committee

Department of Housing and Urban Development
Charles M. Haar
Assistant Secretary for Metropolitan Development

Department of the Interior
Harry Perry
Mineral Resources Research Advisor

Department of Justice
Walter Kiechel, Jr.
Assistant Chief
General Litigation Section
Land and Natural Resources Division

Department of Labor
Dr. Leonard R. Linsenmayer
Deputy Director
Bureau of Labor Standards

Department of Transportation
William H. Close
Assistant Director for Environmental Research
Office of Noise Abatement

Department of the Treasury
Gerard M. Brannon
Director
Office of Tax Analysis

Federal Power Commission
F. Stewart Brown
Chief
Bureau of Power

General Services Administration
Thomas E. Crocker
Director
Repair and Improvement Division
Public Buildings Service

National Aeronautics and Space Administration
Major General R. H. Curtin, USAF (Ret.)
Director of Facilities

National Science Foundation
Dr. Eugene W. Bierly
Program Director for Meteorology
Division of Environmental Sciences

Post Office Department
Louis B. Feldman
Chief
Transportation Equipment Branch
Bureau of Research and Engineering

Tennessee Valley Authority
Dr. F. E. Gartrell
Assistant Director of Health

Atomic Energy Commission
Dr. Martin B. Biles
Director
Division of Operational Safety

Veterans Administration
Gerald M. Hollander
Director of Architecture and Engineering
Office of Construction

AIR QUALITY CRITERIA FOR PARTICULATE MATTER

TABLE OF CONTENTS

Chapter *Page*

Preface iii

Introduction xiii

1 Atmospheric Particles: Definitions, Physical Properties, Sources Concentrations 1

2 Effects of Atmospheric Particulate Matter on Solar Radiation and and Climate Near the Ground 33

3 Effects of Atmospheric Particulate Matter on Visibility 47

4 Effects of Atmospheric Particulate Matter on Materials 63

5 Economic Effects of Atmospheric Particulate Matter 77

6 Effects of Atmospheric Particulate Matter on Vegetation 87

7 Social Awareness of Particulate Pollution 97

8 Odors Associated with Atmospheric Particulate Matter 103

9 The Respiratory System: Deposition, Retention, and Clearance of Particulate Matter 109

10 Toxicological Studies of Atmospheric Particulate Matter 127

11 Epidemiological Appraisal of Atmospheric Particulate Matter 145

12 Summary and Conclusions 179

Appendices

A—Symbols 192

B—Abbreviations 193

C—Conversion Factors 194

D—Glossary 195

Author Index 204

Subject Index 208

Acknowledgements 211

INTRODUCTION

Pursuant to authority delegated to the Commissioner of the National Air Pollution Control Administration, *Air Quality Criteria for Particulate Matter* is issued in accordance with Section 107b1 of the Clean Air Act (42 U.S.C. 1857c–2b1).

Air quality criteria are an expression of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects on man and his environment. They are issued to assist the States in developing air quality standards. Air quality criteria are descriptive; that is, they describe the effects that have been observed to occur when the ambient air level of a pollutant has reached or exceeded specific figures for a specific time period. In developing criteria, many factors have to be considered. The chemical and physical characteristics of the pollutants and the techniques available for measuring these characteristics must be considered, along with exposure time, relative humidity, and other conditions of the environment. The criteria must consider the contribution of all such variables to the effects of air pollution on human health, agriculture, materials, visibility, and climate. Further, the individual characteristics of the receptor must be taken into account. Table A, which appears at the end of this introduction, is a listing of the major factors that need to be considered in developing criteria.[1]

Air quality standards are prescriptive. They prescribe pollutant exposures which a political jurisdiction determines should not be exceeded in a specified geographic area, and are used as one of several factors in designing legally enforceable pollutant emission standards.

The particulate matter commonly found dispersed in the atmosphere is composed of a large variety of substances. Some of these—flourides, beryllium, lead, and asbestos, for example—are known to be directly toxic, although not necessarily at levels routinely found in the atmosphere today. There may very well be others whose toxic effects have not yet been recognized. To evaluate fully the effects on health and welfare of the presence of each of these substances in the air requires that they be given individual attention, attention as classes of similar substances, or that they be considered in conjunction with other substances where synergistic effects may occur. Such evaluations will be made at a later time in separate documents.

This document focuses on total particulate matter of the kind normally measured by high-volume sampling methods, by paper-tape sampling methods, and by dustfall collection. Further, this document considers the effects of particulate matter in conjunction with some gaseous materials, such as sulfur dioxide, where important synergistic effects are observed. (Atmospheric sulfur oxides are treated in detail in a companion criteria document: *Air Quality Criteria for Sulfur Oxides.*) No attempt is made in this document to set up dose-response relationships for specific particulate pollutants. Also, the large and diverse contributions of agricultural and forest management operations to air pollution, such as insecticide spraying and slash burning, and the ingestion hazard to animals and man of toxic particulate matter deposited on plant materials, are treated only for a few selected examples; details are beyond the scope of this document.

---

[1] Calvert, S. Statement for air quality criteria hearings held by the Subcommittee on Air and Water Pollution of the U.S. Senate Committee on Public Works. July 30, 1968.

Methods of measuring the effects of particulate matter on meteorological conditions, atmospheric visibility, and materials are documented, as well as is the resulting economic loss. The effects of particulate matter are further considered as they relate to vegetation damage. Public awareness of air pollution and the role of particulate matter in the odor problem are assessed. There is a chapter on the respiratory system, and particulate deposition therein and removal therefrom, necessary to understanding of the final chapters which survey toxicological effects of particulate matter and the epidemiological data for man and animals.

In general, the terminology employed follows usage recommended in the publications style guide of the American Chemical Society. A glossary of terms, list of symbols and abbreviations, list of conversion factors for various units of measurement, author index, and subject index are provided.

The literature has been generally reviewed on a worldwide basis through March 1968. The results and conclusions of foreign investigations are evaluated for their possible application to the air pollution problem in the United States. This document is not intended as a complete, detailed literature review, and it does not cite every published article related to atmospheric particulates. However, the literature has been reviewed thoroughly for information related to the development of criteria, and the document not only summarizes the current scientific knowledge of particulate air pollution, but points up the major deficiencies in that knowledge and the need for further research.

Technological and economic aspects of air pollution control are considered in companion volumes to criteria documents. The best methods available for controlling the sources of particulate air pollution, as well as the costs of applying these methods, are described in: *Control Techniques for Particulate Air Pollutants.*

Table A.—FACTORS TO BE CONSIDERED IN DEVELOPING AIR QUALITY CRITERIA

- Properties of Pollution:
  - Concentration
  - Chemical composition
  - Mineralogical structure
  - Adsorbed gases
  - Coexisting pollutants
  - Physical state of pollutant
    - Solid
    - Liquid
    - Gaseous
  - Rate of transfer to receptor domain
- Measurement Methods:
  - Hi-Vol sampler
  - Spot-tape sampler
  - Dust fall bucket (rate of deposition)
  - Condensation nuclei counter
  - Impinger (liquid filled)
  - Cascade impactor
  - Electrostatic precipitator
  - Light scattering meter
  - Chemical analysis
  - Gas analysis (non-adsorbing)
  - Adsorbed gas analysis
  - Light scattering or attenuation
    - (Ringleman or visibility observation)
  - Colored suspension
  - Nucleation of precipitation
  - Stabilization of fog
  - Odor
  - Taste
- Exposure Parameters:
  - Duration
  - Concomitant conditions, such as
    - Temperature
    - Pressure
    - Humidity
- Characteristics of Receptor:
  - Physical characteristics
  - Individual susceptibility
  - State of health
  - Rate and site of transfer to receptor
- Responses:
  - Effects on health (diagnosable effects, latent effects, and effects predisposing the organism to disease):
    - Human health
    - Animal health
    - Plant health
  - Effects on human comfort
  - Soiling
  - Other objectionable surface deposition
  - Corrosion of materials
  - Deterioration of materials
  - Effects on atmospheric properties
  - Effects on radiation and temperature

Chapter 1

ATMOSPHERIC PARTICLES: DEFINITIONS, PHYSICAL PROPERTIES, SOURCES, AND CONCENTRATIONS

Table of Contents

Page
A. INTRODUCTION 5
B. PROPERTIES OF ATMOSPHERIC PARTICULATE MATTER 8
  1. Surface Properties 8
  2. Motion 9
  3. Optical Properties 9
C. CHEMICAL REACTIONS OF ATMOSPHERIC PARTICULATE MATTER 10
D. SOURCES OF ATMOSPHERIC PARTICULATE MATTER 10
E. ATMOSPHERIC PARTICULATE MATTER IN URBAN AREAS 11
  1. Suspended Particulate Matter 11
  2. Dustfall 16
F. SAMPLING AND ANALYSIS OF ATMOSPHERIC PARTICULATE MATTER 17
  1. Particles Larger than 10μ 17
  2. Particles 0.1μ to 10μ 19
    a. Tape Samplers for Suspended Particulate Matter 20
    b. High-Volume Samplers for Suspended Particulate Matter 22
  3. Particles Smaller than 0.1μ 23
G. SIZE, CHEMICAL COMPOSITION, AND SOURCE STRENGTHS OF PARTICULATE MATTER FROM SELECTED EMISSION SOURCES 23
  1. Open Hearth Furnaces 23
    a. Chemical Composition 23
    b. Particle Size 23
  2. Incineration 24
    a. Chemical Composition 24
    b. Particle Size 25
  3. Sulfuric Acid Manufacture: Chamber Process 25
    a. Chemical Composition 25
    b. Particle Size 25
  4. Sulfuric Acid Manufacture: Contact Process 25
    a. Chemical Composition 25
    b. Particle Size 25
  5. Cement Plants 25
    a. Chemical Composition 25
    b. Particle Size 26
  6. Motor Vehicles 26
    a. Chemical Composition 26
    b. Particle Size 26
  7. Fuel Oil Combustion 26
    a. Chemical Composition 26
    b. Particle Size 27
  8. Combustion of Coal 27
    a. Chemical Composition 27
    b. Particle Size 27

Page

H. SUMMARY 27

I. REFERENCES 29

List of Figures

Figure

1–1 Sizes of Atmospheric Particulate Matter 6

1–2 Setting Velocities in Still Air at 0°C and 760 mm Pressure for Particles Having a Density of 1 g/cm^3 as a Function of Particle Diameter 7

1–3 Log-Normal Distribution of Particles Showing Various Average Diameters 7

1–4 Particle Size Distribution of Figure 1–3, Plotted Logarithmically 7

1–5 Cumulative Log—probability Curve for the Distribution of Figure 1–4 8

1–6 Horizontal Elutriator Cut-off Characteristics 17

1–7 Dustfall Data for Six Cities 18

List of Tables

Table

1–1 Emission Inventory of Particulate Material, Tons Per Year 12

1–2 Suspended Particle Concentrations (Geometric Mean of Center City Station) in Urban Areas, 1961 to 1965 13

1–3 Distribution of Selected Cities by Population Class and Particle Concentration, 1957 to 1967 15

1–4 Distribution of Selected Nonurban Monitoring Sites by Category of Urban Proximity, 1957 to 1967 15

1–5 Arithmetic Mean and Maximum Urban Particulate Concentrations in the United States, Biweekly Samplings, 1960 to 1965 16

1–6 Emission Factors for Selected Categories of Uncontrolled Sources of Particulates 24

3/4

Chapter 1

ATMOSPHERIC PARTICLES: DEFINITIONS, PHYSICAL PROPERTIES, SOURCES, AND CONCENTRATIONS

A. INTRODUCTION

Atmospheric particles are chemically a most diverse class of substances; they do, however, have a number of physical properties in common, and for this reason are generally classed in a single category, sometimes referred to as aerosols.

Some workers restrict the concept of particulate matter to the solid phase; this distinction is, however, difficult to make in practice and is probably not proper. Others refer to airborne particles as nuclei because of their role in the nucleation of condensation, especially of liquid or solid water. In this document, the term "particle" is used to mean any dispersed matter, solid or liquid in which the individual aggregates are larger than single small molecules (about 0.0002μ in diameter), but smaller than about 500μ. [One micron (μ) is one-thousandth of a millimeter or one-millionth of a meter.] Particles in this size range have a life-time in the suspended state varying from a few seconds to several months.

Many disciplines are involved in the study of particles, and each appears to have devised its own system of nomenclature to differentiate classes of particles with respect to size, physical state, origin, etc. Periodic, but rather unsuccessful attempts have been made to resolve the confusion.[1] The present document will discuss the several classes of particles by specifying the size or size interval in microns (μ) and, where appropriate, the physical state. Figure 1–1 provides a graphic scheme for relating meteorologic nomenclature for aerosols to the particle sizes. Nonspherical particles may be idealized as spheres which would have the same settling rate, but even so, size designations have frequently been ambiguous. For example, "size" has been taken to mean both diameter and radius. Again some workers interpret size to mean the physical or geometrical size, while others mean some equivalent size based, for example, on optical laws relating the size of aerosol particles to the measured scattering of a light beam. In this document, "size" ordinarily refers to particle diameter or Stokes' diameter as defined below.

Particles larger than about a micron in diameter settle in still air at velocities approximated by Stokes' Law:

$$v = \frac{gd^2\,(\rho 1 - \rho 2)}{18\,n} \qquad (1\text{–}1)$$

where

v is velocity in cm/sec (settling velocity or terminal velocity),
g is the acceleration of gravity in cm/sec^2,
d is particle diameter in cm,
ρ_1 and ρ_2 are the densities of the particle and of air respectively in g/cm^3, and
n is viscosity of air in poise.

The expression is precisely true only for spheres. An upper limit to its applicability is set by the fact that, when a certain settling velocity is reached, the particle generates a significant "wake". A lower limit is reached when the particles become small enough, around 1μ, that air resistance is no longer continuous but is rather the result of individual collisions with air molecules. Under these conditions the particles "slip" between molecules and the Stokes' equation underestimates their falling velocity. Correction factors exist to allow for this behavior, but they need not be given here for the qualitative discussion which follows. The approximate set-

17

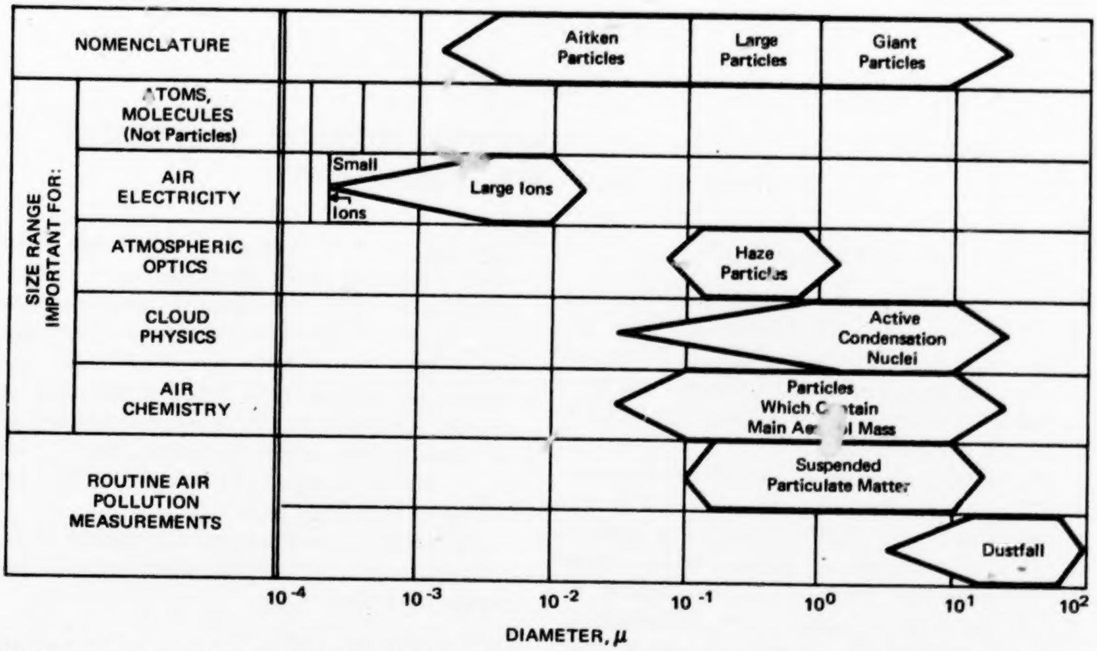

FIGURE 1-1. Sizes of Atmospheric Particulate Matter[2] (The figure shows the ranges of particle size (diameter) of various types of particulate matter which are found in the earth's atmosphere.)

tling velocities in still air at 0°C and 760 mm pressure for particles having a density of 1 g/cm^3 are:

0.1μ, 8×10^{-5} cm/sec; 1μ, 4×10^{-3} cm/sec; 10μ, 0.3 cm/sec; 100μ, 25 cm/sec; and 1,000μ, 390 cm/sec. (See Figure 1-2.)

In the case of a nonspherical particle, substitution of v, g, ρ_1, ρ_2, and n in equation (1-1), Stokes Law, leads to a fictitous diameter, d, which is known as the Stokes or aerodynamic diameter. Unless otherwise stated, the word "diameter," as applied to particles suspended in air or gas, ordinarily means Stokes diameter.

If the density, ρ, of the particles is not known, it may arbitrarily be assigned a value of 1 g/cm^3; in this case d is no longer the "Stokes diameter" but rather the "reduced sedimentation diameter"—that is, the diameter of a spherical particle of unit density having the same terminal fall velocity in still air as the particles in question.

The geometrical diameter of a particle will be smaller than the reduced sedimentation diameter if the particle has a density greater than 1. A few quantitative examples can be given:[3] a 1-μ sphere of lead with a density of approximately 11 has a reduced sedimentation diameter of 3.4μ; a bubble of air in water with an outer diameter of 1μ and a water film thickness of 0.1μ and consequently a density of approximately 0.5 g/cm^3 has a reduced sedimentation diameter of 0.7μ. Nonspherical particles can also be assigned a "diameter" based on their settling rate. A rectangular plate of density 1 g/cm^3, and dimensions $5 \times 5 \times 0.5\mu$, has a reduced sedimentation diameter near 2μ.

Some of the smallest particles may be no more than statistical aggregations of gas molecules which act as a particle at one instant and cease to exist at the next. Solid particles and liquid droplets may be formed in the atmosphere by condensation of a vapor. Solid particles produced by abrasion or grinding are not sphercial and are called dust.

This discussion of size classes must not obscure the fact that there is a continuous spectrum of sizes among the particles in the

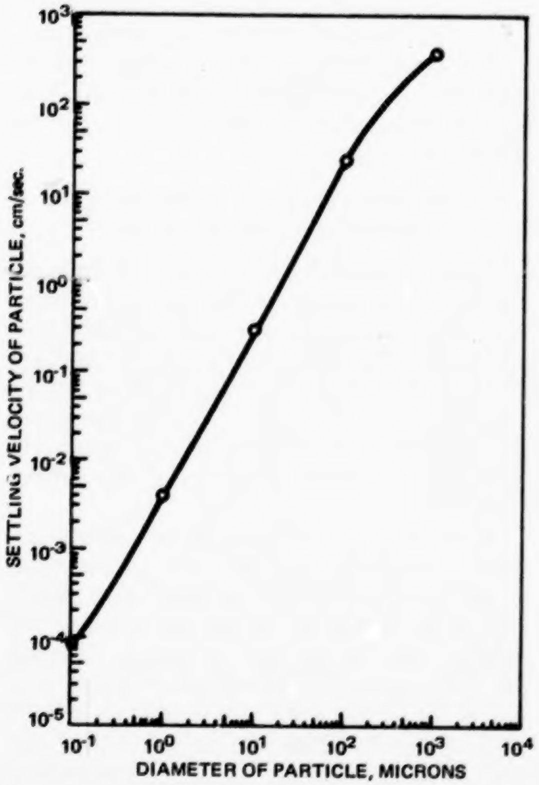

Figure 1-2. Settling Velocities in Still Air at 0° C and 760 mm Pressure for Particles Having a Density of $1g/cm^3$ as a Function of Particle Diameter. (This graph shows that, for spherical particles of unit density suspended in air near sea level, Stokes law applies over a considerable range of particle sizes, where the line is straight, but that correction is required at the extremes where the line begins to curve.)

atmosphere and a corresponding continuous gradation of all their size-dependent properties. The distribution of particle sizes usually encountered approximates closely a log-normal distribution. In this distribution, the familiar symmetrical bell-shaped probability curve appears for a frequency graph plotted against the logarithm of the particle size. In this graph, the ordinate is the number of particles per unit log (particle size) interval. Figures 1-3 and 1-4 show the direct and logarithmic frequency distribution curves for for the log-normal distribution. In practice, a cumulative distribution is plotted on special graph paper with log-probability scales so

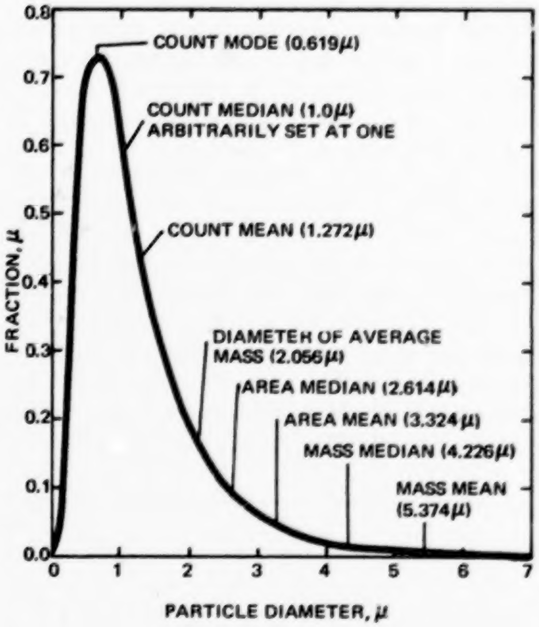

Figure 1-3. Log-normal Distribution of Particles showing Various Average Diameters.[4] (The graph is drawn from probability theory, assuming a count median diameter of 1μ, and shows the numerical values relative to that diameter of several other weighted average diameters discussed in the text.)

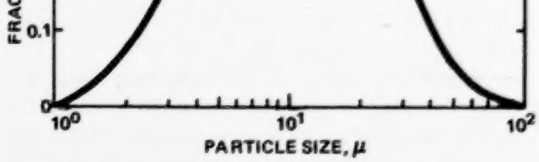

Figure 1-4. Particle Size Distribution of Figure 1-3, Plotted Logarithmically. (If the particle size distribution is log-normal, the graph is symmetrical when plotted logarithmically. The figure should be contrasted with Figure 1-3.)

that a straight line is obtained if the distribution is truly log normal; the best line is fitted either by eye or mathematically. Such a plot is shown in Figure 1–5, and the experimental points give an idea of the extent to which the size distribution of a typical industrial dust approximates to log-normal.

A distribution curve based on an exact mathematical function can be specified in terms of two parameters. In the case of the log-normal distribution, two frequently used defining parameters are (1) the most probable size, which in this distribution is identical with the geometric mean, M_g, and (2) the geometric standard deviation, σg. It can be shown that the 50 percent point of the cumulative graph (Figure 1–5) corresponds to M_g, while the ratio of the 84.1 percent point to the 50 percent point is σ_g.

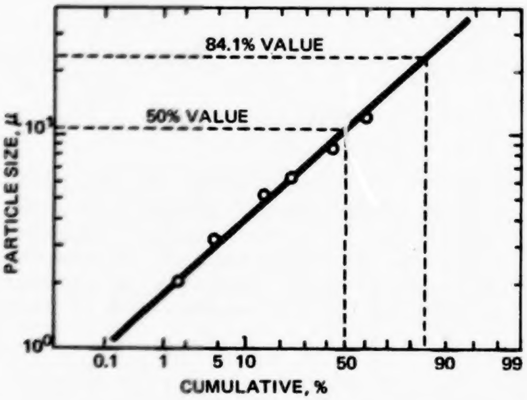

Figure 1–5. Cumulative Log-probability Curve for the Distribution of Figure 1–4. (In a cumulative plot, the experimental points fall on a straight line when the size distribution is log-normal. The 50% point corresponds to the geometric mean, M_g (about 11μ in this case) and the ratio of the 84.1% point to this (about 22/11 or 2, in this case) is the geometric standard deviation, σ_g. In practice, the experimental points usually lie near a straight line, as shown.)

Several averages of particle size may be employed and these may be based on averages of numbers, areas, masses, etc. Some of the most important averages are indicated in Figure 1–3, and the numerical values calculated for the distribution are given relative to a count (number) median diameter of 1μ.[4] The terms used are self-descriptive; half of the quantity (e.g., mass) lies on either side of a *median* diameter, while a *mean* value refers to the diameter of particles possessing a specific weighted average of a quantity. A more extensive mathematical treatment of these quantities is given in books by Cadle [5] and by Drinker and Hatch.[6]

B. PROPERTIES OF ATMOSPHERIC PARTICULATE MATTER

Since particulate matter may consist of such a wide variety of substances, a discussion of chemical properties of the class cannot be specific. Some selected chemical properties of important individual species will be discussed later in connection with analytical methods. Biological properties are most conveniently grouped in terms of the effects produced.

Many physical properties are equally diverse. Diamond particles are harder than gypsum particles of the same size, while hardness in this sense has no meaning for liquids. Shapes are just as varied, although there is a general tendency towards spherical shape with decreasing size as surface energy (which is a minimum for spheres) predominates over crystal energy. Only three general classes of physical properties can reasonably be said to apply to all particulate matter. These properties all involve the interface between the particle and its surroundings, and they are (1) surface properties, (2) motions, and (3) optical properties. A discussion of the properties of aerosols has been given by Corn,[2] and it should be used to extend the remarks offered below.

1. Surface Properties

Surface properties include sorption, nucleation, adhesion, etc. Sorptive behavior can best be understood by considering the impact of individual molecules on the particle surface. If this impact is perfectly elastic and rebound is truly instantaneous, nothing happens. If, however, rebound is delayed or the velocity of rebound is smaller than that of impact, then there will be a local accumulation of the gas on or near the particle surface. If the delay is great, a substantial fraction of the surface may be covered—a phenomenon known as adsorption. If the delay is caused specifically by a chemical interaction between the surface and the gas, the process is known as chemisorption. Absorption refers to the situation in which the gas is dissovled into the particle.

A vapor (i.e., a gas below its critical temperature), present in amounts comparable to its equilibrium vapor pressure, may lead to a deepened sorbed layer, which then takes on the character of a layer of true liquid or solid. If the vapor is supersaturated, a droplet or crystal may grow by further condensation on the sorbed layer. The net result is nucleation, a phenomenon which deserves more consideration. A pure vapor, free of particles, must be highly supersaturated before a condensed phase will form from it, because an energy barrier separates the molecular from the particulate state.

Two like molecules of gas will not generally stick together, and an aggregate of three molecules is still less likely to retain its identity for any length of time. A small aggregate of molecules is therefore unstable. On the other hand, if a particle is split in two, energy is required to create the new surfaces, since the combined surface area of the two fragments is greater than that of the original particle (surface energy increases with a decrease in size). At some point, these two trends of decreasing stability meet at a maximum which corresponds to a certain particle size. If a molecular aggregate can reach this size, then the addition of a single molecule puts it over the energy barrier and it will become more stable by collecting still more molecules. Conversely, the loss of a single molecule from a nucleus of critical size can destroy its stability with the probable result that it will return to the molecular or gaseous state.

The important point is that the critical particle generally contains some tens of molecules which must all come together at once. Unless the vapor concentration is high, this is an improbable event; for some substances, homogeneous nucleation may even require supersaturations of many hundredfold. However, a complete sorbed layer on a particle surface behaves like a drop of the same diameter as the particle, and the energy barrier to producing a droplet is avoided. Since particles are always present in the atmosphere, nucleation on them is of widespread occurrence.

The last of the surface properties of consequence is adhesion. All available evidence suggests that solid particles with diameters less than 1μ (and liquid particles regardless of size) always adhere when they collide with each other or with a larger surface. Other factors being equal, reentrainment or rebound becomes increasingly probable with increasing particle size. Alternatively, the adhesive property can be considered in terms of the surface energy of small particles or in terms of the more complex shear forces acting to dislodge the larger particles.

2. Motion

The second major class of properties common to all particles, regardless of composition, is their mode of motion. Particles with sizes less than 0.1μ undergo large random (Brownian) motions caused by collision with individual molecules. Particles larger than 1μ have significant settling velocities, and their motions can vary significantly from the motion of the air in which they are borne. For particles between 0.1μ and 1μ, settling velocities in still air, though finite, are small compared with air motions. Despite fairly high concentrations, coagulation is somewhat slower as compared with particles smaller than 0.1μ because of decreased Brownian motion. Nevertheless, the operation of this mechanism, together with the processes which generate larger particles and which remove particles from the air, causes the whole population of particulate matter in the air to tend towards a constant size distribution.

Although actual settling times in the atmosphere tend to differ from those computed from Stokes Law, because turbulence tends to offset gravitational fall, the particles larger than 5μ or 10μ are removed to a large extent by gravity and other inertial processes.

3. Optical Properties

The final class of physical properties to be discussed is that of the behavior of particles towards light. This behavior is clearly of importance in effects on visibility, and it is through their optical effects that particles are usually perceived in the atmosphere. Once again, particles in the size range 0.1μ to 1μ exhibit properties showing a transition between two extreme cases.

Particles below 0.1μ are sufficiently small compared to the wavelength of light to obey approximately the same laws of light scattering as molecules do. This so-called Rayleigh scattering varies as the sixth power of the particle diameter and is fairly inconsequential in its effects on visibility. On the other hand, particles very much larger than 1μ are so much larger than the wavelength of visible light that they obey the same laws as macroscopic objects, intercepting or scattering light roughly in proportion to their cross-sectional area. Particles in the intermediate size range obey complex scattering laws set forth by Mie;[7] these laws are beyond the scope of the present discussion. Because the particle dimensions are of the same order of magnitude as the wavelength of visible radiation, interference phenomena play a complicating role, and a given scattering behavior may correspond to several particle sizes. Unfortunately, this is precisely the size range which is most effective in light scattering and thus most needful of study. A more complete discussion of optical effects is given in Chapter 3.

C. CHEMICAL REACTIONS OF ATMOSPHERIC PARTICULATE MATTER

In view of the diverse chemical compositions of particles, it is not possible to make general statements about the chemical reactions of particulate atmospheric pollutants, and the following discussion refers to some specific reaction systems that have been studied. Both particle-gas and particle-particle reactions can occur, but the latter class has been studied to an even lesser extent than the former. Such particle-particle reactions should certainly occur in the size range below 0.1μ where collision between particles is frequent, but, in particles large enough to be readily studied, collisions are relatively infrequent in the atmosphere because of low concentrations. Samples of particles collected on filters may, however, react and subsequent analysis can be very misleading if this fact is not taken into account.

One of the particle-gas systems, the reaction between sulfuric acid mist and ammonia gas, was investigated by Robbins and Cadle.[8] At high humidities the reaction rate was limited by diffusion of ammonia to the mist droplets. At low humidities the droplets were viscous enough to result in diffusion of the reaction product away from the surface of the drops being the rate-determining step. This work shows the effect of accumulation of reaction products, and attempts to explain the role of humidity in a gas-particle reaction.

Goetz and Pueschel[9] reported a study which fully reveals the complexity of even a simplified model of the photochemical air pollution found in Los Angeles. The one clear relationship is that the amount of reaction product deposited on nuclei supplied from the gas phase is proportional to the surface area of the nuclei. The humidity effect is complex and depends upon the amounts and the order of addition of the other substances present. The obvious reactants (olefins, nitrogen dioxide, and sulfur dioxide) differ in action as well. Amounts of sulfur dioxide of the order of 2 ppm depress aerosol formation, while larger amounts (15–16 ppm) increase it. Nitrogen dioxide is more effective if mixed with nuclei before mixing with the olefin.

Interactions between sulfur dioxide and metal oxide aerosols have recently been studied by Smith *et al.*[10] at ambient conditions of temperature and humidity. In measurements that included an adsorption isotherm for sulfur dioxide on dispersed particles, preferential chemisorption on iron oxide and aluminum oxide aerosols was observed at low sulfur dioxide concentrations (up to 2 ppm) followed by multilayered physical adsorption at higher concentrations.

D. SOURCES OF ATMOSPHERIC PARTICULATE MATTER

In a broad sense, particles in the atmosphere are produced by two mechanisms: those in the size range below 1μ arise principally by condensation, while larger particles result from comminution, although there is considerable overlap. For example, Preining *et al.*[11] showed the presence of many particles smaller than 1μ in the spray from a nebulizer, while the formation of very small drops during the rupture of bubbles has been demonstrated.[12] Dry grinding processes are rarely efficient in producing particle sizes below a few microns because of the rapid increase in energy necessary to produce the additional surface.

Combustion is complex in that it may produce four distinct types of particles. These may arise in the following ways:

1. The heat may vaporize material which subsequently condenses to yield particles in the size range between 0.1μ and 1μ,
2. the energy available produces particles of very small size (below 0.1μ); these particles may be of short life as a result of their being simply unstable molecular clusters,
3. mechanical processes may reduce either fuel or ash to particle sizes larger than 1μ and may entrain it,
4. if the fuel is itself an aerosol during combustion, a very fine ash may escape directly, and
5. partial combustion of fossil fuels may result in soot formation.

Particles larger than 10μ frequently result from mechanical processes such as wind erosion, grinding, spraying, etc., although raindrops, snowflakes, hailstones, or sleet are obviously not produced in this way.

The sources of dust are usually apparent. For example, a dustfall sample nearly always contains particles of local soil. Another large fraction will be materials dropped on the ground and pulverized by vehicles, pedestrians or wind action. Although actual soot flocs are increasingly absent as better home heating is used, there may be partially burnt trash from inefficient incinerators. Finally, the process dust characteristic of local industry will be present. In urban locations particles between 1μ and 10μ generally reflect industrial and combustion processes with some local soil also present. In maritime locations, the bulk of the airborne sea salt will be found in particles of this size. The finer process dusts (ash, etc.) also fall into this category. In short, atmospheric particles in the 1-μ to 10-μ range tend to have a composition characteristic of local sources and soil.

As mentioned before, it is difficult to form small particles by size reduction. The class of particles between 0.1μ and 1μ compared with the larger particles therefore tends to contain increasing amounts of condensation products. Products of combustion begin to predominate together with photochemical aerosols. Particles below 0.1μ have not been characterized chemically but the increase over the natural level, characteristic of cities, seems to be entirely the result of combustion.

Table 1–1 shows typical particulate emission source data. Section G–1 gives some typical analytical data on particulate matter by industry source.

E. ATMOSPHERIC PARTICULATE MATTER IN URBAN AREAS

1. Suspended Particulate Matter

The fraction of aerosol mass in the particles below 0.1μ is small and concentrations are normally reported in number per unit volume. Even the cleanest air rarely contains fewer than some hundreds of particles per cubic centimeter, and the particle count in very polluted urban air [17] may reach $10^5/cm^3$.

The bulk of current data on suspended particles generally does not discriminate on the basis of size. Most data come from the National Air Surveillance Network (formerly the National Air Sampling Network.[18, 19] Blifford [20] has applied factorial analysis to these data to show relationships among individual pollutant species. Average suspended particle mass concentrations range from about 10 $\mu g/m^3$ in remote nonurban areas to about 60 $\mu g/m^3$ in near urban locations. In urban areas, averages range from 60 $\mu g/m^3$ to 220 $\mu g/m^3$, depending on the size of the city and its industrial activity. In heavily polluted areas, values of up to 2000 $\mu g/m^3$ have been recorded.

Table 1–2 lists the average suspended particle concentrations for a number of standard metropolitan statistical areas through-

Table 1-1.—EMISSION INVENTORY OF PARTICULATE MATERIAL, TONS PER YEAR.

Source Class	Metropolitan Area							
	New York-New Jersey [13]		Washington [14]		St. Louis [15]		Los Angeles [16]	
	1966		1965–66		1963		1965	
	Tons	Percent	Tons	Percent	Tons	Percent	Tons	Percent
Fuel combustion	134,410	58.1	19,280	55.4	86,800	58.9	8,580	18.8
Power generation	40,042	17.3	9,912	28.5	22,400	15.2	4,825	10.5
Coal	31,722	13.7	-------	-------	22,400	15.2	-------	-------
Anthracite	47	-------	-------	-------	-------	-------	-------	-------
Bituminous	31,675	13.7	9,890	28.4	-------	-------	-------	-------
Fuel Oil	7,593	3.3	22	0.1	-------	-------	-------	-------
Distillate	-------	-------	19	0.1	-------	-------	-------	-------
Residual	7,593	3.3	3	-------	-------	-------	-------	-------
Natural Gas	727	-------	-------	-------	68	-------	-------	-------
Industrial	33,599	14.5	351	1.0	39,000	26.5	730	1.6
Coal	23,442	10.5	135	0.4	37,990	25.8	-------	-------
Anthracite	8,022	3.5	-------	-------	-------	-------	-------	-------
Bituminous	15,420	6.7	135	0.4	-------	-------	-------	-------
Fuel Oil	9,569	4.1	182	0.5	683	0.5	-------	-------
Distillate	1,479	0.6	23	0.1	-------	-------	-------	-------
Residual	8,090	3.5	159	0.5	-------	-------	-------	-------
Natural Gas	588	-------	34	0.1	423	0.3	-------	-------
Domestic	41,073	17.8	3,166	9.1	19,900	13.5	2,425	6.6
Coal	17,767	7.7	735	2.1	18,873	12.8	-------	-------
Anthracite	16,561	7.2	685	2.0	-------	-------	-------	-------
Bituminous	1,206	0.5	50	0.1	-------	-------	-------	-------
Fuel Oil	21,580	9.3	1,839	5.3	671	0.5	-------	-------
Distillate	15,326	6.6	1,154	3.3	-------	-------	-------	-------
Residual	6,254	2.7	685	2.0	-------	-------	-------	-------
Natural Gas	1,726	0.7	592	1.7	354	0.2	-------	-------
Commercial and Government	19,696	8.5	5,851	16.8	5,500	3.7	Included with domestic	
Coal	8,139	3.5	3,891	11.2	5,450	3.7		
Anthracite	4,432	1.9	153	0.4	-------	-------	-------	-------
Bituminous	3,707	1.6	3,738	10.7	-------	-------	-------	-------
Fuel Oil	10,894	4.7	1,814	5.2	34	-------	-------	-------
Distillate	3,281	1.4	661	1.9	-------	-------	-------	-------
Residual	7,613	3.3	1,153	3.3	-------	-------	-------	-------
Natural Gas	663	-------	146	0.4	27	-------	-------	-------
Refuse disposal	41,734	18.0	8,155	23.4	15,800	10.7	365	0.8
Incinerator	-------	-------	-------	-------	1,700	1.2	365	0.8
Open burning	-------	-------	-------	-------	14,100	9.6	-------	-------
Transportation	35,245	15.2	6,245	18.0	7,100	4.8	21,535	47.0
Motor Vehicles	33,761	14.6	5,678	16.3	4,700	3.2	17,155	37.5
Gasoline	22,630	9.8	4,031	11.6	4,100	2.8	16,425	35.9
Diesel	11,131	4.8	1,647	4.7	600	0.4	730	1.6
Aircraft	-------	-------	410	1.2	211	0.1	4,015	8.8
Shipping	1,484 [a]	0.6	-------	-------	670	0.5	365 [b]	0.8
Railroads	-------	-------	157	0.5	1,500	1.0	-------	-------
Industrial Process	19,914	8.6	1,110	3.2	37,500	25.4	13,865	33.5
Asphalt Batching	-------	-------	-------	-------	198	0.1	365	0.8
Asphalt Roofing	-------	-------	-------	-------	NA	-------	1,095	2.4
Cement Plants	-------	-------	-------	-------	3,600	2.4	No plants	
Chemical Plants	-------	-------	-------	-------	NA	-------	2,920	6.8
Coffee Processing	-------	-------	-------	-------	38	-------	Not reported	
Coke Plant	Not reported		Not reported		73	-------	No plants	
Glass and Frit	Not reported		Not reported		NA	-------	730	1.6
Grain Industry	-------	-------	-------	-------	6,695	4.5	Not reported	

See footnotes at end of table.

Table 1-1 (continued).—EMISSION INVENTORY OF PARTICULATE MATERIAL, TONS PER YEAR.

Source Class	Metropolitan Area							
	New York-New Jersey [13]		Washington [14]		St. Louis [15]		Los Angeles [16]	
	1966		1965–66		1963		1965	
	Tons	Percent	Tons	Percent	Tons	Percent	Tons	Percent
Metals	-------	-------	-------	-------	12,433	8.3	2,920	6.4
Ferrous	-------	-------	-------	-------	12,392	8.3	1,460	3.2
Nonferrous	-------	-------	-------	-------	41	-------	1,460	3.2
Solvent Uses [c]	-------	-------	-------	-------	NA	-------	5,470	11.9
Sulfuric Acid Mfg	-------	-------	-------	-------	192	0.1	Not reported	
Superphosphate Mfg	-------	-------	-------	-------	223	0.2	No plants	
Other	-------	-------	-------	-------	14,063	9.5	365	0.8
Total	231,303	100.0	34,790	100.1	147,400	100.0	44,345	100.0

[a] Both aircraft and shipping.

[b] Both shipping and railroads.

[c] Includes chemical plant emissions of solvents.

NA Not available.

Table 1-2.—SUSPENDED PARTICLE CONCENTRATIONS (GEOMETRIC MEAN OF CENTER CITY STATION) IN URBAN AREAS, 1961 TO 1965.

Standard metropolitan statistical area	Total suspended particles		Benzene-soluble organic particles	
	μg/m³	Rank	μg/m³	Rank
Chattanooga	180	1	14.5	2
Chicago-Gary-Hammond-East Chicago	177	2	9.5	19.5
Philadelphia	170	3	10.7	12.5
St. Louis	168	4	12.8	4
Canton	165	5	12.7	5
Pittsburgh	163	6	10.7	12.5
Indianapolis	158	7	12.6	6
Wilmington	154	8	10.2	15
Louisville	152	9	9.6	18
Youngstown	148	10	10.5	14
Denver	147	11	11.7	8.5
Los Angeles-Long Beach	145.5	12	15.5	1
Detroit	143	13	8.4	28
Baltimore	141	14.5	11.0	10
Birmingham	141	14.5	10.9	11
Kansas City	140	16.5	8.9	23
York	140	16.5	8.1	34
New York-Jersey City-Newark-Passaic-Patterson-Clifton	135	18	10.1	16
Akron	134	20	8.3	30.5
Boston	134	20	11.7	8.5
Cleveland	134	20	8.3	30.5
Cincinnati	133	22.5	8.8	25
Milwaukee	133	22.5	7.4	42
Grand Rapids	131	24	7.2	44.5
Nashville	128	25	11.9	7
Syracuse	127	26	9.3	23
Buffalo	126	27.5	6.0	56
Reading	126	27.5	8.8	25
Dayton	123	29	7.5	40.5
Allentown-Bethlehem-Easton	120.5	30	6.8	50
Columbus	113	31.5	7.5	40.5
Memphis	113	31.5	7.6	39

Table 1–2 (continued).—SUSPENDED PARTICLE CONCENTRATIONS (GEOMETRIC MEAN OF CENTER CITY STATION) IN URBAN AREAS, 1961 TO 1965.

Standard metropolitan statistical area	Total suspended particles		Benzene-soluble organic particles	
	μg/m³	Rank	μg/m³	Rank
Portland (Oreg.)	108	34	9.5	19.5
Providence	108	34	17.7	38
Lancaster	108	34	6.8	50
San Jose	105	36.5	14.0	3
Toledo	105	36.5	5.6	58
Hartford	104	38.5	7.1	46
Washington	104	38.5	9.4	21
Rochester	103	40	6.1	55
Utica-Rome	102	41	7.0	47
Houston	101	42	6.8	50
Dallas	99	43	8.8	25
Atlanta	98	44.5	7.8	36.5
Richmond	98	44.5	8.3	30.5
New Haven	97	46	7.3	43
Wichita	96	47	5.2	60
Bridgeport	93	50	7.2	44.5
Flint	93	50	5.3	59
Fort Worth	93	50	7.8	36.5
New Orleans	93	50	9.7	17
Worcester	93	50	8.2	33
Albany-Schenectady-Troy	91.5	53	6.6	52
Minneapolis-St. Paul	90	54	6.5	53
San Diego	89	55	8.5	27
San Francisco-Oakland	80	56	8.0	35
Seattle	77	57	8.3	30.5
Springfield-Holyoke	70	58	7.0	47.5
Greensboro-High Point	60	59	6.3	54
Miami	58	60	5.7	57

out the United States. For the most part, measurements were taken at a single sampling station in the downtown area of the city.

Based on ten years of sampling at approximately 370 sites, the highest seasonal average will exceed the annual mean by 15 to 20 percent. Seasonal averages for the high 2 percent of the sites exceeded the annual mean by 50 percent and the lowest 2 percent exceeded the annual mean by about 5 percent. Individual 24-hour maximum sample concentrations vary widely from the annual mean, and on the average this variation is from 280 to 300 percent. Variations as high as 700 percent of the annual mean are found for the high two percent of the samples. Sunday and holiday data are usually 15 to 20 percent below weekday concentrations. Table 1–3 shows the relation of population class of urban areas to particle concentration for the period 1958–1967, while Table 1–4 shows the frequency distribution of particle concentration in nonurban areas for the same period.

Particle concentrations in air have both diurnal and annual (seasonal) cycles which for most cities are generally predictable in shape. A city with cold winters will experience a seasonal maximum in midwinter as a result of increased fuel use for space heating. A daily maximum in the morning, probably between 6 and 8 o'clock, usually relates to a combination of meteorological factors and an increase in the strength of sources of particulates, including the automobile traffic.

A reflection of the effect of the strength of various sources can also be seen in the previously mentioned lower weekend and holiday versus weekday concentrations.

In cities where photochemical pollution

Table 1–3. DISTRIBUTION OF SELECTED CITIES BY POPULATION CLASS AND PARTICLE CONCENTRATION, 1957 TO 1967.

[Avg. particle concentration $\mu g/m^3$]

Population class	<40	40 to 59	60 to 79	80 to 99	100 to 119	120 to 139	140 to 159	160 to 179	180 to 199	>200	Total cities in table	Total cities in U.S.A.
>3 million	----	----	----	----	----	----	1	----	1	----	2	2
1–3 million	----	----	----	----	----	----	2	1	----	----	3	3
0.7–1 million	----	----	1	----	2	----	4	----	----	----	7	7
400–700,000	----	----	----	4	5	6	1	1	1	----	18	19
100–400,000	----	3	7	30	24	17	12	3	2	1	99	100
50–100,000	----	2	20	28	16	12	6	5	1	3	93	180
25–50,000	----	5	24	12	12	10	2	1	2	3	71	--------
10–25,000	----	7	18	19	9	5	2	3	1	----	64	[a] 5,453
<10,000	1	5	7	15	11	2	1	2	----	----	44	--------
Total urban	1	22	77	108	79	52	31	16	8	7	401	--------

[a] Incorporated and unincorporated areas with population over 2,500.

Table 1–4.—DISTRIBUTION OF SELECTED NON-URBAN MONITORING SITES BY CATEGORY OF URBAN PROXIMITY, 1957 TO 1967.[21]

Category	Average particle concentrations, $\mu g/m^3$				Total
	<20	20–39	40–59	60–79	
Near urban [a]	-----	1	3	1	5
Intermediate [b]	-----	5	6	-----	11
Remote [c]	4	5	-----	-----	9
Total nonurban	4	11	9	1	25

[a] Near urban—although located in unsettled areas, pollutant levels at these stations clearly indicate influence from nearby urban areas. All of these stations are located near the northeast coast "population corridor."

[b] Intermediate—distant from large urban centers, some agricultural activity, pollutant levels suggest that some influence from human activity is possible.

[c] Remote—minimum of human activity, negligible agriculture, sites are frequently in state or national forest preserve or park areas.

predominates, the maximum in concentration of particles in the range from 0.1μ to 1μ may come around noon, after the sun has had an opportunity to cause photochemical reaction. Under these conditions, the highest concentration of particles below 0.1μ will come earlier, and there may be no clear trend for larger particles.

The above concentrations generally relate to samples taken in the center-city commercial district. This portion of the community will generally not show annual average concentrations as high as those found in various industrial areas; however, they are among the higher area concentrations in a community. Annual concentrations in nearby suburban residential areas generally will be about one-half of that found in center city.

Particulate air pollution is not only source- and location-dependent but is also a function of meteorological factors causing a variation in the natural ventilation of a community. Air pollution episodes are characterized by minimum natural ventilation, and particulate concentrations at such times may rise dramatically as indicated by the following examples: during the November-December, 1962, episode in the Eastern United States, particulate concentrations in several communities rose to two-to-three times normal;[22] during the Thanksgiving 1966 episode, again in the Eastern United States, particulate concentrations increased by about a factor of two over mean autumn levels. In fact, maximum citywide average concentrations in Philadelphia, Worcester, and Boston exceeded maximum concentrations recorded for an autumn period since 1961 at the National Air Surveillance Network (NASN) stations.[23]

Table 1–5 gives concentrations of certain specific contaminants found in total sus-

Table 1–5. ARITHMETIC MEAN AND MAXIMUM URBAN PARTICULATE CONCENTRATIONS IN THE UNITED STATES, BIWEEKLYY SAMPLINGS, 1960 TO 1965.[19]

Pollutant	Number of stations	Concentrations μg/m³	
		Arith. average [a]	Maximum
Suspended particulates	291	105	1254
Fractions:			
Benzene-soluble organics	218	6.8	([b])
Nitrates	96	2.6	39.7
Sulfates	96	10.6	101.2
Ammonium	56	1.3	75.5
Antimony	35	0.001	0.160
Arsenic	133	0.02	([b])
Beryllium	100	<0.0005	0.010
Bismuth	35	<0.0005	0.064
Cadmium	35	0.002	0.420
Chromium	103	0.015	0.330
Cobalt	35	<0.0005	0.060
Copper	103	0.09	10.00
Iron	104	1.58	22.00
Lead	104	0.79	8.60
Manganese	103	0.10	9.98
Molybdenum	35	<0.005	0.78
Nickel	103	0.034	0.460
Tin	85	0.02	0.50
Titanium	104	0.04	1.10
Vanadium	99	0.050	2.200
Zinc	99	0.67	58.00
Gross beta radioactivity	323	(0.8 pCi/m³)	(12.4 pCi/m³)

[a] Arithmetic averages are presented to permit comparable expression of averages derived from quarterly composite samples; as such they are not directly comparable to geometric means calculated for previous years' data. The geometric mean for all urban stations during 1964–65 was 90 μg/m³, for the nonurban stations, 28 μg/m³.

[b] No individual sample analyses performed.

pended particulate matter. Certain sub-fractional contaminants found in total particulates may be related to community parameters; for example, average ambient vanadium concentrations correlate well with the kind of residual oil used, iron and manganese correlate and are attributed to their joint emission from ferromanganese blast furnaces, and annual gasoline sales correspond with the average lead fraction of suspended particulates. Similarly, sulfates correlate with particulates in those communities which derive large amounts of energy from the higher sulfurous fuels.[24]

2. Dustfall

Dustfall is the usual index of particles in the size range greater than 10μ, and it has mainly been reported in short tons per square mile per month, arrived at by extrapolation from a jar a few inches in diameter to a square mile. Metric units are preferable and the current trend is clearly in their favor. Typical values for cities are 0.35 to 3.5 mg/cm²-month (10 to 100 tons/mile²-month), while values approaching 70 mg/cm²-month (2000 tons/mile²-month) have been measured near especially offensive sources.

A search for scientific interpretations or correlations of dustfall data has been unsuccessful. There is no question that, within a given city, dustfall tends to increase with the intensity of human activity. Furthermore, dustfall measurements are certainly valuable in obtaining evidence against major sources of dust. However, trying to extract detailed information from small fluctuations in dustfall appears to be an exercise in futility. Dustfall is complex, being affected by the number of unvegetated vacant lots, vehicular traffic, uncontrolled heavy industry, and wind velocity. Dustiness of the environment is an obvious nuisance and a component of the economic cost of pollution.

F. SAMPLING AND ANALYSIS OF ATMOSPHERIC PARTICULATE MATTER

1. Particles Larger Than 10μ

Particles larger than 10μ exist in the atmosphere in very low numerical concentrations. The high concentrations that are sometimes found in ducts or in work spaces are the province of industrial hygiene and are not considered here.

Since the largest particles have appreciable settling velocities and impact readily at low velocities, they are usually determined gravimetrically following collection by deposition in a dustfall jar.[25] Although a cylindrical jar might be expected to collect the equivalent of the dust content of an air column of its own diameter extending to the top of the atmosphere, in fact the aerodynamic effects of the jar edges, of the mounting brackets for the jar, and of adjacent structures tend to complicate the collection pattern. Only relative significance may be attached to the resulting data, and only then if conditions are carefully standardized.[26–28] (There is a legend of a city which decreased its reported dustfall by half in a single year by changing the height of its dustfall jars from 8 to 20 feet above ground level.) There is no definitive study of the effect on measured dustfall of the height of the collector above ground.

Gruber [29] has successfully used an adhesive coating on the outside of cylindrical containers to ascertain the wind direction corresponding to maximum dust content of the air. This often permits identification of major dust sources. Evaluation is visual. European practice favors flat adhesive surfaces placed horizontally as dust collectors.[30] The advantages are not apparent, and analogous studies using greased microscope slides for pollen collection have shown them to be highly variable in collection efficiency.[31]

Cyclonic collectors have been employed in combination with high-volume samplers for the selective sampling of particles.[32] While such collectors can remove virtually all particles above 5μ, they also remove a significant amount of smaller particles. During an investigation of atmospheric protein, a small cyclone separator was used ahead of a high-volume sampler. Particles exhausted through the cyclone outlet were collected on a filter of the high-volume sampler. The samples collected on the filter of the combination unit averaged about one-half the weight of those collected at the same time on the filter of a high-volume sampler with no cyclone attached.

A few studies [32–34] have used long horizontal tunnels as fractional elutriators to determine particle size distributions. The elutriator acts as a prefilter for the removal of larger-sized particles in a manner similar to the cyclone high-volume sampler combination. Both the cyclone and the elutriator, operating on aerodynamic principles, have a graded selectivity (Figure 1–6) rather than a sharp cut-off point at a specific particle size. The size range of the particles which penetrate the elutriator but are retained on the filter at the outlet duct depends on the airflow rate through the system (Figure 1–6).

Other methods for the selective removal of larger (nonrespirable) particles have been described by Lippmann and Harris [36] and by Roesler.[37]

The first stage of most cascade impactors [38] collects particles larger than about 5 to 10μ. Since, except in very dusty atmospheres, the mass mean diameter is smaller than this, collections on the first stage will be meager unless the sampling time is set specifically to

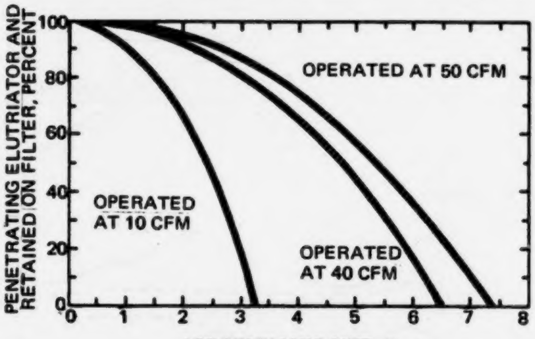

FIGURE 1–6. Horizontal Elutriator Cut-off Characteristics.[35] (This graph shows that the elutriator collects all particles larger than 3½μ diameter when operated at 10 cfm, but at 50 cfm some particles as large as 7μ diameter escape.)

give ample material. Adhesion of such large particles is poor (Section B–1), so an adhesive may be necessary to avoid bounce-off or reentrainment.

A variation is a single-stage impactor with size discrimination developed by Dessens.[39] He used a coarse slit followed by a shaped channel to induce turbulent deposition of particles along a microscope slide with a size gradation from larger to smaller.

It appears, therefore, that no presently used technique for the concentration measurement of particles larger than about 10μ is superior to a properly installed dustfall jar; this method is also the least expensive. However, the jar lacks time resolution since it must usually be exposed for two weeks to a month to obtain a significant sample. Dustfall jars should be more widely standardized, and more study is needed of alternative means of sampling the largest particles in the atmosphere. Chapter 11 shows some correlations of health effects with air pollution, using dustfall as an index of air pollution.

Any collection technique can provide a sample for subsequent analysis, although the adhesives used in many of the methods described can, unless carefully chosen, interfere severely with characterization of the particles. The standard techniques used to analyze dustfall samples generally reveal which elements are somewhere in the sample without giving any information as to which particles contain which elements or what compounds these elements represent. Nevertheless, such general chemical composition data are often helpful. One simple type of chemical characterization which gives this sort of information for particles larger than 10μ is morphological identification under the microscope. Although this may be applied to smaller particles as well, it is most effective in the largest size range. McCrone[40] has published a photomicrographic atlas of dust components which should permit recognition of up to 90 percent of the particles above 10μ in a typical urban sample. In the hands of an experienced microscopist, this technique is one of the most potent tools in dust analysis. X-ray diffraction techniques will identify chemical compounds present rather than merely the elements.

Dustfall levels have decreased in most cities (Figure 1–7) and there is a trend

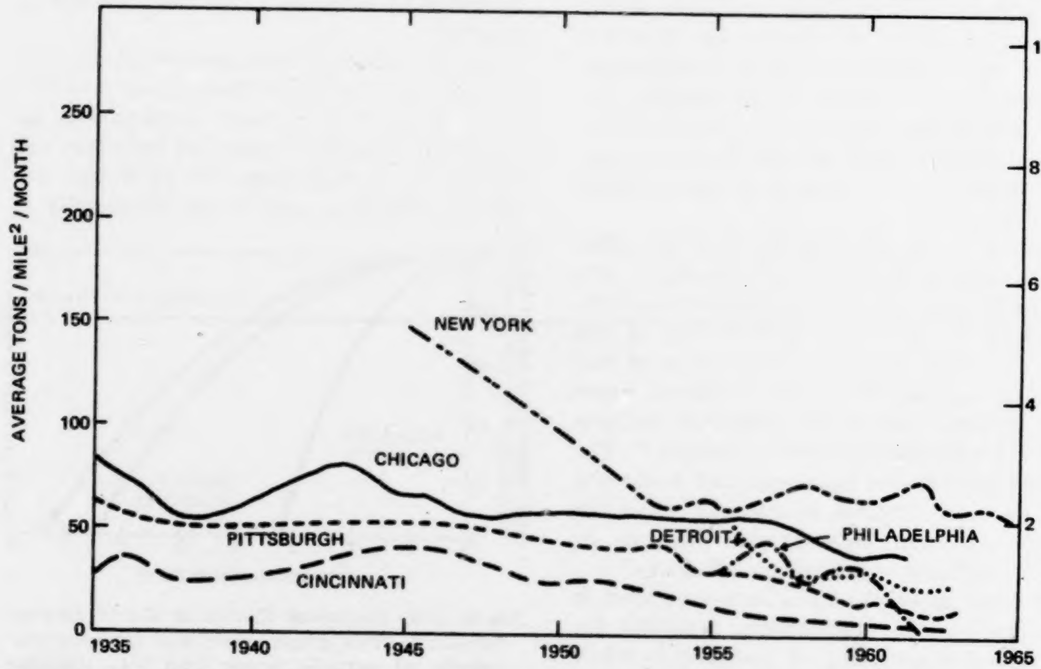

Figure 1–7. Dustfall Data for Six Cities. (This graph is from a U.S. Government publication, but the original source of the data is unknown.)

toward abandoning routine dustfall measurements, as they may no longer be indicative of pollution levels. This viewpoint is defensible, although, since excessive dustfall is one of the most noticeable nuisances consequent upon air pollution, there is public pressure to abate high dust emissions (Chapter 7).

2. Particles 0.1μ to 10μ

A single group of sampling and analysis methods generally serves for the size range from 0.1μ to 10μ. This size range includes both the bulk of the particulate mass and a large fraction of the numbers. The preponderant optical effects also arise from particles in this portion of the size spectrum, and most of the estimation methods not involving collection are optical. Collection and analysis techniques in this size range have been reviewed by Lodge [41, 42] and discussed in two chapters of the 1968 treatise, *Air Pollution*, edited by Stern.[43, 44]

The simplest optical technique involves use of a photometer developed in its present form by Volz [45] for determining air turbidity. A simple photocell is pointed at the sun through a series of small apertures and a glass filter peaking at a wavelength of 500μ. An attached sight and spirit level allow measurement of sun angle, which corrects the reading for air layer thickness along the path between the instrument and the sun. A nomogram may then be used to obtain the turbidity coefficient. Greater accuracy, if warranted, may be achieved by using a computer; a simple program has been developed for the calculation.[46] McCormick [47] found that turbidity measurements from the bottom and from the top of a high building gave, by difference, a reasonable estimate of the concentration of the intervening particulate matter. This is the cheapest and simplest technique, although it cannot measure continuously nor can it be used at night or during cloudy weather.

Next in complexity is the "nephelometer" described by Charlson.[48] This device measures light scattered by particles suspended in a defined volume of air. It is illuminated by a flash tube placed so that nearly the total solid angle from full forward to direct back-scattering occurs. Integral scattering is recorded and, in the absence of a powerful nearby particle source, is very nearly a linear function of mass concentration. A disadvantage of the method is that a constant particle-size distribution and composition must be assumed.

Other light-scattering instruments include total forward-scattering and right-angle-scattering photometers as well as instruments which count and size individual particles.[49] The latter consist of:

1. A sampling system which dilutes the sample stream with purified air until only one particle at a time is likely to be in the sensitive portion of the device,
2. a light source and optics to illuminate a small volume (a few mm^3 at most) at a defined angle,
3. a phototube (usually a multiplier) sensitive enough to detect the individual flashes of light as particles pass the illuminated volume, and
4. a pulse height analyzer and counting electronics.

A number of other principles for size analysis of particles without collection were surveyed during World War II and the immediate postwar years, but none seem to have warranted commercial exploitation except one device to measure mobility in an electric field. This latter technique is more applicable to smaller particles and will be discussed in that connection (see Section G-3).

Cascade impactors [38] have been found to be useful devices for simultaneously collecting and classifying particles throughout most of this size range, thereby yielding considerable information [50–53] on the urban aerosol size distribution of selected chemical components including sulfate, lead, and other metals. Similar information has been obtained by use of a helical-channel centrifuge [54–56] as a classifier.

Goetz [57] has described a single-stage moving-slide impactor with some novel features. It is part of a system which includes mirror-surfaced collecting slides and incident dark-field microphotometry which is particularly well adapted to physical characterization of particles from roughly 0.2μ to 2μ. Information on coalescence tendency and heat lability is easily obtained.

Spurny[58] has studied the application of recently developed filter material to particle research. The filter is a plane film of polycarbonate which is exposed to fission fragments and then treated to remove the radiation damage. A number of pore sizes are available; the pores are uniform, straight, and circular in cross section. The plane upper surface is ideal for both optical and electron microscopy (the latter after replication), and the filtration characteristics are excellent. The uniformity of the material commends its use for gravimetric purposes and for "smoke shade" determination. The only evident disadvantage at the moment is its price.

Frank and Lodge[59] have described the use of electron microscopy for morphological identification of several species of particles smaller than 1μ. Although the technique is not so broadly applicable as is optical microscopy of dusts, it may permit some analyses, including the identification of sulfuric acid droplets below 1μ.

In view of the extensive literature involving filter tape and high-volume samplers, some further discussion of these devices is in order, especially since air quality criteria must ultimately relate to accurate measurements. Both samplers are inexpensive and durable, and both provide data which have stringent limitations that are not always understood.

a. Tape Samplers for Suspended Particulate Matter

The tape sampler most widely used in the United States has been the AISI (American Iron and Steel Institute) sampler developed by Hemeon and his colleagues.[60] Other versions exist, but all are alike in function. A series of portions of filter paper, usually successive areas of a paper tape, are positioned so as to be clamped between an intake tube and a vacuum connection. Air is drawn through the filter for a selected time, usually one to four hours, and a new portion of tape is then moved into position and sampling is resumed.

The fundamental basis of evaluating samples is optical, although a few nonoptical methods have been studied. The visual color of the spots may be compared with a standard gray scale. The reflectance may be measured photometrically. The transmittance of light through both filter and deposit may be compared with transmittance through a clean portion of the filter. Visual and reflectance measurements determine the blackness of the deposit, while transmittance measures a function of all particles collected, and under some circumstances measurements made by the two techniques may not be identical. For example, a gram of magnesium oxide smoke collected in a single spot would not be visibly gray and might even increase reflectance, but would transmit no light at all. Urban particulate matter is not, however, pure magnesium oxide, and, in fact, the three measures (visual, reflectance, and transmittance) generally correlate fairly well in the short run. Over longer periods, the introduction of new sources and the removal of old ones may be expected to change the composition of the particles enough to cause divergence of the different techniques. For example, if a residential area abruptly converts from coal heating to gas, the virtual disappearance of soot will have an enormous effect on reflectance which may not appear so strikingly in transmittance.

Transmittance is by no means a unique function of the total concentration of particulate matter. Stalker *et al.*[61] compared transmittance and particle concentrations in different parts of Nashville, Tennessee. The slopes of the regression lines varied by a factor greater than three, and at some locations no correlations appeared to exist between the two measurements.

Additional complications arise in three ways. First, none of the methods of measurement exhibit a linear relationship between the quantity determined and the number of particles collected. Reflectance changes will depend on whether later deposits are retained on the surface of, or penetrate deeply into, the layers first deposited. Measurements of transmittance are similarly dependent on whether an added increment simply adds thickness to the collected deposit or fills pores and gaps, thus increasing the bulk density but not the thickness of the layer.

Second, the rate of sampling is not constant but depends on the amount of material already collected and the structure of the deposit.[62] Hence the assumption of a constant sampling rate will be seriously in error, especially for heavy deposits. Averaging initial and final sampling rates, or recording the sampling rate continuously, represent improvements at the cost of greatly increased attention to the instrument, or of increased complexity and expense.

Third, reflectance methods lose all discrimination beyond a certain deposit density. Once the filter is covered with particulate matter, only the exposed surface of the deposit affects the reflectance; the reflectance then become a measure of the composition, but not the amount of material collected. This problem is recognized, but not solved, in a proposed European standard method.[63]

Transmittance, which is most widely measured in the United States, is normally converted into units of coh's per thousand linear feet of air passing through the filter. Coh stands for "coefficient of haze." A coh unit is defined as that quantity of light-scattering solids (on the filter) which produces an optical density equivalent of 0.01 when measured by light transmission. Optical density is defined as the common (decadic) logarithm of the opacity (inverse of fractional transmission). Thus if one-fifth of the incident light is transmitted through the paper, as compared with clean paper, the opacity is five. A coh measurement is routinely reduced to coh per 1,000 linear feet of air passing through the filter tape by dividing the unreduced coh value by the number of thousands of feet actually drawn in the test.

For reflectance, a RUDS test is made by measuring the percentage reflectance of the filter tape and similarly reducing the measurement to 1,000 linear feet of air. Reflectance of clean paper tape is the reference standard, set at 100 on the reflectometer. RUDS is an acronym for "reflectance unit of dirt shade."

Reflectance measurements are used in various European countries for calculating ambient air concentrations of "smoke" or "dark suspended matter."[63] The darkness of the filter stain is not considered proportional to total particle concentration in the air but to the concentration of "dark suspended matter" resulting primarily from combustion. The darkness of the stain, as measured by a reflectometer, is converted to surface concentration of smoke by means of a calibration curve. The derived surface concentration is then translated into an ambient concentration using the relationship:

$$C = \frac{SA}{V} \tag{1-2}$$

where C = the concentration of smoke in the air ($\mu g/m^3$),
S = the derived surface concentration ($\mu g/cm^2$),
A = the area of the filter stain (cm^2), and
V = the volume of air sampled (m^3).

Experimental work on the form of the calibration curve for deriving surface concentrations has been carried out in France, the Netherlands, and the United Kingdom. The Working Party on Methods of Measuring Air Pollution and Survey Techniques of the Organization for Economic Cooperation and Development has proposed a Standard International Calibration Curve (the mean of the curves developed in the three countries listed above) as well as a standard sampling and measurement procedure.[63]

The use of the conversion curves to make international comparisons is potentially dangerous. The method has the disadvantage of credibility; that is, it is too easy to overlook the arbitrary nature of the units, and to believe that actual airborne particle concentrations have been measured. This is of concern for those areas of the United States for which air quality standards already exist; in most cases the standards include separate values for "airborne particulate matter" expressed in gravimetric units, and for "atmospheric soiling" determined by transmittance or reflectance measurements on deposits of the sort described here.

For many purposes, the International Standard Calibration Curve will be found inconvenient. It is the empirical product of a complex physical phenomenon (light absorption and scattering) and an arbitrary instrument response. The curve is not a mathematical one, and hence computer reduction of data is made awkward.

Still other conversion formulae have been suggested by Kemeny[64] in South Africa, by Ellison[65] in England, and by Sullivan[66] in Australia. These workers, following Clark,[67] have attempted to convert their readings mathematically into units of milligrams per cubic meter of "smoke" in the air. As Sanderson and Katz[68] have correctly stated, "There is considerable doubt whether a truly satisfactory expression exists for the translation of optical density readings into units related to smoke and haze concentrations." The results of Stalker *et al.*,[61] previously mentioned, suggest that this may be an understatement. The method yields only an index of and not a measurement of absolute concentrations of suspended particulate matter.

Notwithstanding its limitations, the tape sampler is cheap, simple, and rugged, and it will certainly continue to be used. It should, however, be used with the knowledge that its measurements, in whatever units, are arbitrary and artificial and without absolute meaning. Their *relative* values can be useful if the samplers, as well as their locations, their installation, and the technique of measurement, are rigorously standardized. Furthermore, it must be kept in mind that long-term trends may reflect changes in composition as well as in amount of airborne particles.

b. *High-Volume Samplers for Suspended Particulate Matter*

The original high-volume sampler consisted of the motor and blower of a tank-type vacuum cleaner, suitably enclosed and fitted with a holder for flat filter paper in place of a dust bag. Present versions are more refined, but little different in concept. The use of a blower necessitates a filter of large area and low air resistance, and also makes the sampling rate very dependent on the mass of material collected.

Current samplers[19] are generally exposed inside a case which places the filter surface horizontal, facing upward, under a roof which keeps out rain and snow, and generally prevents collection of particles larger than about 100μ. Filters are felts of glass or synthetic organic fiber. Since the fibers are substantially less than 1μ in diameter, these filters are highly efficient despite their open structure and consequent low resistance to airflow. Samples are normally collected for 24 hours and sampling rates are measured at the beginning and end of the period.

Since the filters are weighed before use, it is possible to determine the weight of collected material if one standardizes the weighing conditions, optimally at 25°C and at relative humidities below 50 percent. Thereafter, samples may be extracted, heated, or incinerated, and determination can be made of organic content, carbon, minerals or any other suitable and/or interesting fraction, element, or substance. The collected sample is the particulate content of approximately 2,000 m^3 of air, and is large enough for nearly any sort of analysis, although care must be used in interpreting the data. There are a few studies on interactions between collected species, loss through volatilization, and similar problems. The performance characteristics of high-volume samplers today are quite well understood, though reactions on sampler filters are not.

Many analyses are plagued by the lack of a universally applicable filter material. Glass fiber filters are convenient for determining total particle concentration. However, the very fine glass fibers are water and acid soluble, and the glass contains significant amounts of a large number of metals, as well as sulfate, silicate, and other anions. Hence inorganic analyses are performed over a background from the filter which is by no means constant. Polystyrene fiber filters can be made extremely low in inorganic content, but are virtually useless for organic analysis. Membrane filters are very useful in special applications, e.g., when alkaline metals are to be determined.

Suspended particle concentrations, determined by high-volume samplers in urban areas, are shown in Table 1–2. The column listing "Benzene soluble organic particles" is a measure of the organic particulate matter in the total sample. Much of this material is derived from the incomplete combustion of fuels. The data on organics may be further analyzed for polycyclic aromatic hydrocarbon content; a possible significance of these compounds in carcinogenesis is discused in Chapter 10.

3. Particles Smaller Than 0.1μ

Several techniques have been used systematically for the characterization of particles smaller than 0.1μ.

1. Saturation of the air and subsequent rapid expansion to cause a high supersaturation: the resulting droplet count is assumed equal to the total particle concentration. Successively smaller degrees of supersaturation presumably activate only larger particles to act as nuclei. Size spectra may be generated in this way, although the results do not necessarily agree with those of other methods. Most of the available data were obtained by this technique.[69]
2. Passage of the air through a long narrow channel: The smallest particles will be removed most rapidly by diffusion, and the extend of the effect can be calculated.[70] Differential condensation nuclei counts after different diffusion lengths permit determination of a size spectrum.
3. Measurement of the mobility of charged particles in an electric field: It is necessary to assume or to compute the efficiency of electrical charging of these smallest particles to derive numbers and effective sizes. Orr and his coworkers [71] used this method to study changes in the size of hygroscopic particles with relative humidity, and Whitby [72] has set up a facility for obtaining count-size distributions of particles in air, using a series of instruments with overlapping ranges.
4. Electron microscopic techniques have been used to obtain particle counts as well as information on the size and morphology of these small particles.

Of these methods only the electrical mobility separator has been used [71] for chemical characterization of particles below 0.1μ, but little analytical information is now available. The electron microscopic methods of Frank and Lodge,[59] and the earlier work of Tufts and Lodge,[73] provide some insight into the chemical composition of the particles, although even in the most favorable cases, the authors were able to account for the composition of less than half of the particles seen.

G. SIZE, CHEMICAL COMPOSITION, AND SOURCE STRENGTHS OF PARTICULATE MATTER FROM SELECTED EMISSION SOURCES

A listing of source strengths is given in Table 1–6. Composition of particles and, where available, data on particle size distribution from various sources follow.

1. Open-Hearth Furnaces

a. Chemical Composition

Analysis [74] of particulate emissions from a 200-ton oxygen-lanced open-hearth furnace, a composite sample for all process stages, indicates the following chemical composition:

Compound	*Percent*
Fe_2O_3	89.1
FeO	1.9
SiO_2	0.9
Al_2O_3	0.5
MnO	0.6
Alkalis	1.4
P_2O_5	0.5
S	0.4

Fluorides may be present in open-hearth furnace particulate emissions if fluorspar fluxes or fluoride-containing ores are used. While fluoride emissions are generally insignificant, problems have been reported in the vicinity of at least one plant which uses fluorspar fluxes and one which uses an ore with a high fluoride content.[75]

b. Particle Size

By number, the majority of particles emitted by an open-hearth furnace are below 0.1μ in diameter.[76] Size analysis [74] of a composite sample over the entire hearth indicated the following distribution:

Diameter (μ)	*Weight percent less than stated size*
2	20
5	46
10	68
20	85
40	93

2. Incineration

a. Chemical Composition

Analysis of emissions from municipal incinerators in Los Angeles [77] indicated 20 percent by weight of the discharge to be condensable and approximately 5 to 15 percent of the condensate to be sulfuric acid. The remaining 80 percent was particulate matter containing silicon, lead, aluminum, calcium, iron, and traces of other elements.

Particulate samples from the stack effluent of municipal incinerators in Milwaukee,[78] ashed and subjected to spectographic and

Table 1-6.—EMISSION FACTORS FOR SELECTED CATEGORIES OF UNCONTROLLED SOURCES OF PARTICULATES.[a]

Emission source	Emission factor
Natural gas combustion:	
Power plants	15 lb/million ft^3 of gas burned
Industrial boilers	18 lb/million ft^3 of gas burned
Domestic and commercial furnaces	19 lb/million ft^3 of gas burned
Distillate oil combustion:	
Industrial and commercial furnaces	15 lb/thousand gallons of oil burned
Domestic furnaces	8 lb/thousand gallons of oil burned
Residual oil combustion:	
Power plants	10 lb/thousand gallons of oil burned
Industrial and commercial furnaces	23 lb/thousand gallons of oil burned
Coal combustion:	
Cyclone furnaces	2X (ash percent) lb/ton of coal burned
Other pulverized coal-fired furnaces	13–17X (ash percent) lb/ton of coal burned
Spreader stokers	13X (ash percent) lb/ton of coal burned
Other stokers	2–5X (ash percent) lb/ton of coal burned
Incineration:	
Municipal incinerator (multiple chamber)	17 lb/ton of refuse burned
Commercial incinerator (multiple chamber)	3 lb/ton of refuse burned
Commercial incinerator (single chamber)	10 lb/ton of refuse burned
Flue-fed incinerator	28 lb/ton of refuse burned
Domestic incinerator (gas-fired)	15 lb/ton of refuse burned
Open burning of municipal refuse	16 lb/ton of refuse burned
Motor vehicles:	
Gasoline-powered engines	12 lb/thousand gallons of gasoline burned
Diesel-powered engines	110 lb/thousand gallons of diesel fuel burned
Grey iron cupola furnaces	17.4 lb/ton of metal charged
Cement manufacturing	38 lb/barrel of cement produced
Kraft pulp mills:	
Smelt tank	20 lb/ton of dried pulp produced
Lime kiln	94 lb/ton of dried pulp produced
Recovery furnaces [b]	150 lb/ton of dried pulp produced
Sulfuric acid manufacturing	0.3–7.5 lb. acid mist/ton of acid produced
Steel manufacturing:	
Open-hearth furnaces	1.5–20 lb/ton of steel produced
Electric arc furnaces	15 lb/ton of metal charged

[a] For more detailed data, consult "Control Techniques for Particulate Air Pollutants," U.S. Department of Health, Education, and Welfare, Dec. 1968

[b] With primary stack gas scrubber

wet chemistry analysis, had the following composition:

SPECTOGRAPHIC ANALYSIS

Elements reported in percent of ashed material

Element	*Percent*
Calcium	10+
Silicon	5+
Sodium	1–10
Nickel	1–10
Aluminum	1–10
Zinc	1–10
Magnesium	1–10
Titanium	0.5–5.0
Iron	0.5–5.0
Barium	0.1–1.0

Small amounts (less than 1 percent) of manganese, chromium, copper, vanadium, tin, silver, boron, beryllium, and lead were present.

WET CHEMICAL ANALYSIS

	Percent
Phosphorus	1.46
Silicate	......
Phosphates	0.88
Nitrates	0.62
Sulfates	5.0
Chlorides	0.02

Analysis of fly ash collected at three New York[79] incinerators and of that emitted from their stacks showed the following chemical composition:

	Weight percent	
	Collected	*Emitted*
Silicon as SiO_2	49.5	36.3
Aluminum as Al_2O_3	22.9	25.7
Iron as Fe_2O_3	6.3	7.1
Calcium as CaO	8.8	8.8
Magnesium as MgO	2.2	2.8
Sodium as Na_2O Potassium as K_2O	6.0	10.4
Titanium as TiO_2	1.3	0.9
Sulfur as SO_3	3.0	8.0

b. Particle Size

Analysis of the particulate emissions from the Los Angeles municipal incinerators indicated 30 percent (by weight) of the particles were less than 5μ in diameter. Particle size analysis of the samples collected at the Milwaukee[78] incinerators showed the following distribution:

Diameter (μ)	*Weight percent less than stated size*
5	6.0
10	20.5
20	47.2
30	68.7
44	89.2

3. Sulfuric Acid Manufacture: Chamber Process

a. Chemical Composition

Acid mist emissions contain sulfuric acid and dissolved nitrogen oxides. The nitrogen oxides constitute approximately 10 percent by weight of total acid mist emissions.

b. Particle Size

The weight percentage of acid mist particles less than 3μ in diameter found in samples from two chamber acid plants, one using molten dark sulfur and one using solid sulfur, were 10.1 percent and 3.5 percent respectively.

4. Sulfuric Acid Manufacture: Contact Process[80]

a. Chemical Composition

Discharge gases contain sulfuric acid mist as well as unabsorbed sulfur trioxide, which converts to acid mist upon reaching the atmosphere. Trace amounts of nitrogen oxides may arise if the fuel used in the process contains nitrogenous matter.

b. Particle Size

In plants where particle size has been determined, the weight percentage of particles 3μ or less in diameter leaving the absorber unit ahead of any mist recovery equipment ranged from 7.5 percent to 95 percent. The mean percentage was 63.5.

When oleum is produced, the proportion of acid mist particles smaller than 3μ in diameter increases. In one plant, the percentage rose from 9.5 percent to 54 percent.

5. Cement Plants

a. Chemical Composition

Chemical analysis of the raw kiln feed dust and the kiln dust from the precipitator outlet of portland cement plants in the Le-high Valley, Pennsylvania, region showed the following composition:

	Weight percent	
Compound*	Raw kiln feed dust (Average for three types of cement)	Dust from precipitator outlet (Average of three samples)
CaO	......	40.9
$CaCO_3$	75.9	......
SiO_2	13.4	18.8
Al_2O_3	3.7	7.1
Fe_2O_3	2.1	9.6
MgO	......	2.5
Na_2O	......	1.1
K_2O	......	7.3
MnO	......	0.2
TiO_2	......	0.1
CuO	......	Trace
Ignition Loss	......	12.7

*No determination of sulfur made.

b. Particle Size

Examples of the distribution of particle sizes in cement plant raw kiln feed and kiln emissions are indicated below:

Diameter (μ)	Raw kiln feed weight percent less than stated size	Kiln emissions weight percent less than stated size	
	Ref. 81	*Ref. 81*[a]	*Ref. 1*
60	81.4	......	97–100
50	73.1	......	95–100
40	63.8	96.5	85–95
30	53.3	92.9	70–90
20	41.5	84.6	50–70
10	23.5	56.3	30–55
5	10.8	15.5	20–40
2.5	......	......	10–35

[a] Average of two samples

6. Motor Vehicles

a. Chemical Composition

Particles contained in vehicle exhaust include lead compounds, carbon particles, motor oil, and nonvolatile reaction products formed from motor oil in the combustion zone. The reaction products include high molecular weight olefins, carbonyl compounds (aldehydes and ketones), and free acids. Lead particles in the exhaust are principally in the from of PbClBr, the α and β forms of $NH_4Cl \cdot 2PbClBr$ and $2NH_4Cl \cdot PbClBr$. Particulates discharged through the blowby consist almost entirely of unchanged lubricating oil.[82]

b. Particle Size

Analysis[83] of diluted exhaust from automobiles operated at crusing conditions showed a particle concentration of 40 to 52μg per liter of exhaust. From 62 to 80 percent of the particulate mass consisted of particles with aerodynamic diameters below 2μ at unit density. The lead content of particulate emissions averaged about 40 percent and appeared to be independent of particle size. Measurements[84] with undiluted auto exhaust indicate that about 90 percent by weight of exhaust lead is contained in particles with diameters below 0.5μ.

7. Fuel Oil Combustion

a. Chemical Composition

The probable constituents[85] of fly ash from oil combustion have been identified as Al_2O_3, $Al_2(SO_4)_3$, CaO, $CaSO_4$, Fe_2O_3, $Fe_2(SO_4)_3$, MgO, $MgSO_4$, NiO, $NiSO_4$ SiO_2, Na_2SO_4, $NaHSO_4$, $Na_2S_2O_7$, V_2O_3, $V_2O_4 \cdot V_2O_5$, ZnO, $ZnSO_4$, $Na_2C \cdot V_2O_5$, $2Na_2O \cdot V_2O_5$, $3Na_2O \cdot V_2O_5$, $2NiO \cdot V_2O_5$, $3NiO \cdot V_2O_5$, $Fe_2O_3 \cdot V_2O_5$, $Fe_2O_3 \cdot 2V_2O_5$, $Na_2O \cdot V_2O_4 \cdot 5V_2O_5$ and $5Na_2O \cdot V_2O_4 \cdot 11V_2O_5$.

Analysis[86] of fly ash from a plant using residual oil produced the following percentage composition:

Element	Test A Total solids from burning PS 400 oil (Collected in a laboratory Electrical precipitator at 230°F) *(Weight percent)*	Test B Total solids from burning 4° API oil (Collected in a glass filter sock at 300°F) *(Weight percent)*
Carbon	58.1[a]	18.1
Ether Soluble	2.3	4.4
Ash (900°C)	17.4	51.2
Sulfates as SO_3 (Including H_2SO_4)	17.5	25.0
Iron as Fe_2O_3	3.1	3.7
Nickel as NiO	1.8	13.2
Vanadium as V_2O_3	2.5	4.7
Silicon as SiO_2	0.6	9.7
Aluminum as Al_2O_3	1.6	14.9
Sodium as Na_2O	0.9	3.0

[a] May include some hydrogen

Less than 1 percent of the following elements or compounds was present: Cl, NO_3, CrO_2, Co_2O_3, BaO, MgO, PbO, CaO, CuO, TiO_2, MoO_2, B_2O_3, MnO_2, ZnO, P_2O_5, SrO, TiO.

b. Particle Size

A literature survey [87] of the size distribution of particles emitted by large oil-burning units gave the following results:

SIZE DISTRIBUTION
(Percent by number)

0μ to 1μ	*1μ to 2μ*	*2μ to 5μ*	*5+μ*	*Largest size*
48.4	28.8	16.7	6.1	15μ
64.2	18.8	10.0	7.0	15μ
93.5	3.2	2.0	1.3	20μ
94.8	2.2	1.5	1.0	20μ

One reference indicated 47 percent, by weight, was less than 3μ diameter.

8. Combustion of Coal [88]

a. Chemical Composition

The following ranges in chemical composition were indicated by analysis of fly ash emissions from a variety of coal combustion units. The figures are the extreme values found in four investigations, each of which reports wide ranges also.

Compound	*Percentage of fly ash*
Carbon, C	0.37–36.2[a]
Iron (as Fe_2O_3 or Fe_3O_4)	2.0 –26.8
Magnesium (as MgO)	0.06– 4.77
Calcium (as CaO)	0.12–14.73
Aluminum (as Al_2O_3)	9.81–58.4
Sulfur (as SO_3)	0.12–24.33
Titanium (as TiO_2)	0 – 2.8
Carbonate (as CO_3)	0 – 2.6
Silicon (as SiO_2)	17.3 –63.6
Phosphorus (as P_2O_5)	0.07–47.2
Potassium (as K_2O)	2.8 – 3.0
Sodium (as Na_2O)	0.2 – 0.9
Undetermined	0.08–18.9

[a] Ignition loss

b. Particle Size

Estimated particle size distributions [88] for four broad classifications of combustion equipment are listed below. All distributions represent the size of the particles leaving the boiler or furnace before any control equipment. The distributions reported for all four equipment classifications ranged widely; those shown in the table are considered "typical."

WEIGHT PERCENT LESS THAN STATED SIZE

Particle Size (μ)	Pulverized Fuel Fired Furnace	Cyclone Furnace	Spreader Stoker-Fired Furnace	Stoker-Fired (Other-than Spreader)
10	30	76	10	7
20	50	83	20	15
40	70	90	37	26
60	80	92	47	36
80	85	94	54	43
100	90	95	60	50
200	96	97	—	66

H. SUMMARY

Aerodispersed solid and liquid particles constitute a significant fraction of the pollutants found in urban atmospheres. Such particulate matter vary greatly in chemical composition and consist of multimolecular assemblies that may range in complexity from salt crystals and acid droplets to heterogeneous liquid and solid aggregates and living cells. In this document, a particle is any dispersed matter, solid or liquid, in which the individual aggregates are larger than single molecules (about 0.0002μ), but smaller than about 500μ diameter.

Atmospheric particles have size-dependent dynamic, optical, and electrical properties, and are characterized by such surface activities as sorption, nucleation, and adhesion. Particles in the size range below 0.1μ display a behavior similar to that of molecules and are characterized by large random motions caused by collisions with gas molecules.

In addition, they frequently collide with each other and form larger aggregates. Particles larger than 1μ have significant settling velocities; their motions deviate from the motion of the air in which they are borne, and their rates of coagulation into larger aggregates are low. The aerodynamic behavior of particles with diameters from 0.1μ to 1μ is transitional between these two regimes. Particles larger than 10μ have rapid settling velocities and therefore remain in the air for relatively short durations. The size range between 0.1μ and 10μ accounts for the bulk of the particulate mass in the atmosphere.

Particles below 0.1μ obey the same laws of light scattering as molecules do and their effects on visibility are inconsequential. Particles very much larger than 1μ obey the same optical laws as macroscopic objects, intercepting or scattering light roughly in proportion to their cross-sectional area. Particles between 0.1μ and a few microns obey the very complex scattering laws set forth by Mie. This is the particle size range that is most effective in scattering light. (See Chapter 3.)

Particles in the atmosphere can be said to originate by two types of mechanism. Small particles in the size range below 1μ arise principally by condensation and combustion, while the larger particles with the exception of rain, snow, hail, and sleet, result from comminution. Although the chemical composition of particles below 0.1μ diameter has not been widely studied, the increase over natural levels of particles in this size range seems to be entirely due to combustion. Combustion products and photochemical aerosols make up a large fraction of the particles in the range of 0.1-μ to 1-μ diameter. Particles between 1μ and 10μ generally include local soil, fine dusts emitted by industry and, at maritime locations, airborne sea salt. Industrial sources of particulate matter include municipal incineration, cement plants, steel mills, sulfuric acid manufacturing, industrial furnaces, kraft pulp mills, and others. Particles larger than 10μ diameter frequently result from mechanical processes such as highway construction, wind erosion, grinding, spraying, etc., and include material that is dropped on the ground and pulverized by vehicles and pedestrians.

Dustfall measurements provide a rough index of those particles which readily settle out of the air. Typical values encountered in urban areas range from 0.35 mg/cm^2-month to 3.5 mg/cm^2-month (10 tons/mi^2-month to 100 tons/mi^2-month) while values approaching 70 mg/cm^2-month (2000 tons/mi^2-month) have been measured close to very severe sources. Levels of dustfall have apparently declined in cities, and dustfall measurement is probably not useful as an index of overall particulate levels. Nevertheless, dustfall itself constitutes a nuisance, and its measurement provides some indication of urban dirtiness.

A number of methods are available to measure the mass concentration of suspended particles. Optical techniques, such as the sun photometer, the integrating nephelometer, and light-scattering counters, provide an indication of particle concentrations in the size range from 0.1μ to 10μ. Two of the most common instruments for measuring mass concentrations are spot samplers and high-volume samplers. In the former, air is drawn through an exposed portion of a paper tape; then the tape is moved to expose another spot. The spots that result on the tape are evaluated optically by measuring their light reflectance or transmittance. Although the spot sampler is cheap, simple, and rugged, its use is better suited to the determination of relative rather than absolute mass concentrations. High-volume samplers employ a blower which sends air through a special filter over a specified time period. By weighing the material collected by the filter, the mass concentration can be readily determined; chemical analyses also can be carried out. High-volume sampling is the method of choice for measuring particulate levels. As with any other point-sampling method, the location of the sampling instrument is very critical, and data for an entire city should not be based on a single sample located at a single place.

Most of the data on suspended particulates come from the National Air Surveillance Network (NASN), which employs the high-volume sampler. NASN currently consists of about 200 urban and 30 nonurban stations, and it is supplemented by State and local networks. Based on these data, annual geometric mean concentrations of suspended particulate matter range from 60 $\mu g/m^3$ to about 200 $\mu g/m^3$ in urban areas. The maximum average concentrations for 24-hour periods is about 3 times the annual mean with values of 7 times the mean occurring in about 2 percent of the communities. In general, mean particulate concentrations correlate with urban population class, but there is a wide range of concentrations within each urban population class, and many smaller communities have higher concentrations than larger ones. In nonurban areas, typical geometric mean concentrations range between 10 $\mu g/m^3$ and 60 $\mu g/m^3$.

I. REFERENCES

1. Kreichelt, T. E., Kemnitz, D. A., and Cuffe, S. T. "Atmospheric Emissions from the Manufacture of Portland Cement." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio. PHS–Pub–999–AP–17, 1967.
2. Corn, M. "Nonviable Particles in the Air." In: Air Pollution, Chapt. 3, Vol. 1, 2nd edition, A. C. Stern (ed.), Academic Press, New York. 1968, pp. 47–94.
3. Goetz, A. "Parameters for Biocolloidal Matter in the Atmosphere." Proc. Atmos. Biol. Conf., 1965, pp. 79–97.
4. "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract." Task Group on Lung Dynamics for Committee II of the International Radiological Protection Commission, Chairman, P. E. Morrow. Health Physics, Vol. 12, pp. 173–207, 1966.
5. Cadle, R. D. "Particle Size." Reinhold, New York, 1965, 390 pp.
6. Drinker, P. and Hatch, T F. "Industrial Dust, Hygenic Significance, Measurement and Control." 2nd edition, McGraw-Hill, 1954, 401 pp.
7. Mie. G. "Beitrage zur Optik trüber Modien, Speziell Kolloidaler Metallosungen." Ann. Physik, Vol. 25, pp. 377–455, 1908.
8. Robbins, R. C. and Cadle, R. D. "Kinetics of the Reaction Between Gaseous Ammonia and Sulfuric Acid Droplets in an Aerosol." J. Phys. Chem., Vol. 62, pp. 469–471, 1958.
9. Goetz, A. and Pueschel, R. "Basic Mechanisms of Photochemical Aerosol Formation." Atmos. Environ., Vol. 1, pp. 287–306, 1967.
10. Smith, B. M., Wagman, J., and Fish, B. R. "Interaction of Airborne Particles with Gases." Preprint. (Presented at the Symposium on Colloid and Surface Chemistry in Air and Water Pollution, 156th National Meeting, American Chemical Society, Atlantic City, New Jersey, September 11, 1968.) (Also to be submitted for publication in Environmental Science and Technology.)
11. Preining, O., Sheesley, D., and Djordjevic, N. "The Size Distribution of Aerosols Produced by Air Blast Nebulization." J. Colloid Interface Sci., Vol. 23, pp. 458–561, 1967.
12. Day, J. A. "Small Droplets from Rupturing Air-Bubble Films." J. Rech. Atmospheriques, Vol. 1, pp. 191–196, 1963.
13. "New York-New Jersey Air Pollution Abatement Activity, Phase II—Particulate Matter." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio, December 1967.
14. "Washington, D.C. Metropolitan Area Air Pollution Abatement Activity." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio, 1967.
15. Venezia, R. and Ozolins, G. "Air Pollutant Emission Inventory. Interstate Air Pollution Study, Phase II Project Report." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio, 1966.
16. Lemke, E. E., Shaffer, N. R., and Verssen, J. A. "Summary of Air Pollution Data for Los Angeles County." Air Pollution Control District, Los Angeles, California, 1965.
17. Junge, C. E. "Air Chemistry and Radioactivity." Academic Press, New York, 1963.
18. "Air Quality Data, 1964–1965." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, 1966, pp. 1–2.
19. "Air Pollution Measurement of the National Air Sampling Network, 1957-1961." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, 1962, pp. 6–8.
20. Blifford, I. H. and Meeker, G. O. "A Factor Analysis Model of Large Scale Pollution." Atmos. Environ., Vol. 1, pp. 147–157, 1967.
21. "National Air Surveillance Networks, Air Quality Data, 1967." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio (in preparation).
22. "The November-December 1962 Air Pollution Episode in the Eastern United States." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, PHS–Pub–999–AP–7, 1964.
23. "Thanksgiving 1966 Air Pollution Episode in the Eastern United States." U.S. Dept. of Health, Education, and Welfare, National Air Pollution Control Administration, Durham, North Carolina, PHS–Pub–AP–45, 1968.
24. McMullen, T. B., Fensterstock, J. C., Faoro, R. B., and Smith, R. "Air Quality and Characteristic Community Parameters." J. Air Pollution Control Assoc., Vol. 18, pp. 545–549, 1968.
25. "Standard Method for Collection and Analysis of Dustfall." In: 1966 Book of ASTM Standards, Part 23, Industrial Water; Atmospheric Analysis, American Society for Testing and Materials, Philadelphia, Pennsylvania, pp. 785–788. Report ASTM D1 739–62.

26. Nadel, J. A. "Dust Retention Efficiencies of Dustfall Collectors." J. Air Pollution Control Assoc., Vol. 8, pp. 35–38, 1958.
27. Sanderson, H. P., Brandt, P., and Katz, M. "A Study of Dustfall on the Basis of Replicated Latin Square Arrangements of Various Types of Collectors." J. Air Pollution Control Assoc., Vol. 13, pp. 461–466, 1963.
28. Sensenbaugh, J. D. and Hemeon, W. C. L. "Variables in Monthly Dustfall Measurements." Am. Soc. Testing Mater. Proc., Vol. 53, pp. 1160–1165, 1953.
29. Gruber, C. W. and Jutze, G. A. "The Use of Sticky Paper in an Air Pollution Monitoring Program." J. Air Pollution Control Assoc., Vol. 7, pp. 115–117, 1957.
30. Diem, M. and Jurksch, G. "Vergleichsmessungen des Staubgehalts der Luft nach Niederschlags- und Konzentrationsmethoden." Staub, Vol. 21, pp. 345–355, 1961.
31. Shelden, J. M. and Hewson, E. W. "Atmospheric Pollution by Aeroallergens." Progress Report No. 3, 1 May 1958 to 30 June 1959, University of Michigan Research Institute, Ann Arbor, Michigan, June 1959, pp. 65–70.
32. Hamilton, R. J. and Walton, W. H. "The Selective Sampling of Respirable Dust." In: Inhaled Particles and Vapours, 1st edition, C. N. Davies (ed.), Pergamon Press, London, England, 1961, pp. 465–481.
33. Slater, R. W. "Low Cost Measurement of Air Pollution." Bull. Univ. Missouri, Vol. 64, pp. 49–53, 1963. (See also Slater, R. W., same title, Ind. Water Wastes, Vol. 8, pp. 30–33, 1963.)
34. Stoeber, W. "Zur Bestimmung von Teilchengrössenverteilungen mit einem Horizontal-Elutriator." Staub, Vol. 24, pp. 221–223, 1964.
35. Robson, C. D. and Foster, K. E. "Evaluation of Air Particulate Sampling Equipment." Am. Ind. Hyg. Assoc. J., Vol. 23, pp. 404–410, 1962.
36. Lippmann, M. and Harris, W. B. "Size-Selective Samplers for Estimating 'Respirable' Dust Concentrations." Health Physics, Vol. 8, pp. 155–163, 1962.
37. Roesler, J. R. "Applications of Polyurethane Foam Filter for Respirable Dust Separation." J. Air Pollution Control Assoc., Vol. 16, pp. 30–34, 1966.
38. Andersen, A. A. "A Sampler for Respiratory Health Hazard Assessment." Am. Ind. Hyg. Assoc. J., Vol. 27, pp. 160–165, 1966.
39. Dessens, J. "Un capteur classeur de particules a lame unique." Bull. Obs. Puy de Dome, 1–13, 1961.
40. McCrone, W. C., Draftz, R. G., Ronald, G., and Delly, J. G. "The Particle Atlas." Science Publishers, Inc., Ann Arbor, Michigan, 1967, 406 pp.
41. Lodge, J. P. "Identification of Aerosols." In: Advances in Geophysics, Vol. 9, H. E. Landsberg and J. Van Miegham (eds.), Academic Press, New York, 1962, pp. 97–130.
42. Lodge, J. P. and Pate, J. B. "Air Analysis and Atmospheric Pollution." In: Treatise on Analytical Chemistry, Part III, Vol. 2, Sect. D, I. M. Kolthoff and P. J. Elving (eds.), Interscience, New York. (In press, 1968).
43. West, P. W. "Chemical Analysis of Inorganic Particulate Pollutant." In: Air Pollution, Chapter 19, Vol. 2, 2nd edition, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 147–185.
44. Hoffman, P. and Wynder, E. L. "Chemical Analysis and Carcinogenic Bioassay of Organic Particulate Pollutants." In: Air Pollution, Chapt. 20, Vol. 2, 2nd edition, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 187–247.
45. Volz, F. "Photometer mit Selen-Photoelement zur spektralen Messung der Sonnenstrahlung und zur Bestimmung der Wellenlängenabhangigkeit der Dunsttrübung." Arch. Meteorol., Geophys., Bioklimatol., Ser. B, Vol. 10, pp. 100–131, 1959.
46. Fischer, W. H. and Swartztrauber, P. Unpublished manuscript, 1968.
47. McCormick, R. A. and Baulch, D. M. "The Variation with Height of the Dust Loading over a City as Determined from the Atmospheric Turbidity." J. Air Pollution Control Assoc., Vol. 12, pp. 492–496, 1962.
48. Charlson, R. J., Horvath, H., and Pueschel, R. F. "The Direct Measurement of Atmospheric Light Scattering Coefficient for Studies of Visibility and Air Pollution." Atmos. Environ., Vol. 1, pp. 469–478, 1967.
49. Gucker, F. T., O'Konski, C. T., Pickard, H. B., and Pitts, J. N., Jr. "A Photoelectric Counter for Colloidal Particles." J. Am. Chem. Soc., Vol. 69, pp. 2422–2431, 1947.
50. Roesler, J. R., Stevenson, H. J. R., and Nader, J. S. "Size Distribution of Sulfate Aerosols in the Ambient Air." J. Air Pollution Control Assoc., Vol. 15, pp. 576–579, 1965.
51. Corn, M. and DeMaio, L. "Particulate Sulfates in Pittsburgh Air." J. Air Pollution Control Assoc., Vol. 15, pp. 26–30, 1965.
52. Wagman, J., Lee, R. E., Jr., and Axt, C. J. "Influence of Some Atmospheric Variables on the Concentration and Particle Size Distribution of Sulfate in Urban Air." Atmos. Environ., Vol. 1, pp. 479–489, 1967.
53. Lee, R. E., Jr., Patterson, R. K., and Wagman, J. "Particle Size Distribution of Metal Components in Urban Air." Environ. Sci. Technol., Vol. 2, pp. 288–290, 1968.
54. Robinson, E. and Ludwig, F. L. "Size Distributions of Atmospheric Lead Aerosols." Stanford Research Institute, Menlo Park, California, Final Report SRI Project PA–4788, 1964.
55. Ludwig, F. L. and Robinson, E. "Size Distribution of Sulfur-Containing Compounds in Urban Aerosols." J. Colloid Sci., Vol. 20, pp. 571–584, 1965.
56. Ludwig, F. L. and Robinson, E. "Variations in the Size Distributions of Sulfur-Containing Compounds in Urban Aerosols." Atmos. Environ., Vol. 2, pp. 13–23, 1968.

57. Goetz, A. "A New Impaction Device for the Determination of Aerosol Levels and Particle Constitution." Preprint. (Presented at the 152nd Annual Meeting, American Chemical Society, New York, September 11–16, 1966.)

58. Spurny, K. and Lodge, J. P. "Die Aerosolfiltration mit Hilfe der Kernporenfilter." Staub, Vol. 28, pp. 179–186, 1968.

59. Frank, E. R. and Lodge, J. P. "Morphological Identification of Airborne Particles with the Electron Microscope." J. Microscopie, Vol. 4, pp. 449–456, 1967.

60. Hemeon, W. C. L., Haines, G. F., Jr., and Ide, H. M. "Determination of Haze and Smoke Concentration by Filter Paper Samplers." Air Repair, Vol. 3, pp. 22–28, 1953.

61. Stalker, W. W., Dickerson, R. C., and Kramer, G. D. "Atmospheric Sulfur Dioxide and Particulate Matter, a Comparison of Methods of Measurement." Am. Ind. Hyg. Assoc. J., Vol. 24, pp. 68–79, 1963.

62. Park, J. C., Keagy, D. M., and Stalker, W. W. "Developments in the Use of the A. I. S. I. Automatic Smoke Sampler." J. Air Pollution Control Assoc., Vol. 10, pp. 303–306, 1960.

63. "Methods of Measuring Air Pollution. Report of the Working Party on Methods of Measuring Air Pollution and Survey Techniques." Organization for Economic Cooperation and Development, Paris, France, 1964, 94 pp.

64. Kemeny, E. "The Determination of Gravimetric Pollution Concentrations by Means of Filter Paper." J. Air Pollution Control Assoc., Vol. 12, pp. 278–281, 1962.

65. Ellison, J. M. "Die Schätzung der Konzentration teilchenformiger Luftverunreinigungen durch Bestimmung des Verschmutzungsgrades von Filtertapieren." Staub, Vol. 28, pp. 240–246, 1968.

66. Sullivan, J. L. "The Calibration of Smoke Density." J. Air Pollution Control Assoc., Vol. 12, pp. 474–478, 1962.

67. Clark, J. G. "Calibration of Dr. Owen's Apparatus for the Estimation of Suspended Solid Pollution in the Atmosphere. Report on Observations in the Year 1966–67." Advisory Committee on Air Pollution.

68. Sanderson, H. P. and Katz, M. "The Optical Evaluation of Smokes or Particulate Matter Collected on Filter Paper." J. Air Pollution Control Assoc., Vol. 13, pp. 476–482, 1963.

69. Rich, T. A., Pollak, L. W., and Metnieks, A. L. "Estimation of Average Size of Submicron Particles from the Number of All and Uncharged Particles." Geofis. Pura Appl., Vol. 44, pp. 233–241, 1959.

70. Megaw, W. J. "Recent Research on Small Ions and Aitken Nuclei." J. Rech. Atmospheriques, Vol. 2, pp. 53–68, 1966.

71. Orr, C., Hurd, F. K., Hendrix, W. P., and Junge, C. E. "The Behavior of Condensation Nuclei under Changing Humidities." J. Meteorol., Vol. 15, pp. 240–242, 1958.

72. Whitby, K. T. and Clark, W. E. "Electric Aerosol Particle Connecting and Size Distribution Measuring System for the 0.015 to 1μ Size Range." Tellus, Vol. 18, pp. 573–586, 1966.

73. Tufts, B. J. and Lodge, J. P. "Chemical Identification of Halide and Sulfate in Submicron Particles." Anal. Chem., Vol. 30, pp. 300–303, 1958.

74. Strauss, W. "Cleaning Waste Gases from Open Hearth Steel Processes." Chem. Process Eng., Vol. 41, pp. 339–343, 331, 1960.

75. Purvance, W. T. "Atmospheric Pollution Control." Chem. Process Eng., Vol. 55, pp. 49–53, 1959.

76. Silverman, L. "Technical Aspects of High Temperature Gas Cleaning for Steel Making Processes." Air Repair, Vol. 4, pp. 189–196, 321, 1955.

77. Chass, R. L. and Rose, A. H. "Discharge from Municipal Incinerators." Preprint. (Presented at the 46th Annual Meeting, Air Pollution Control Association, 1953.)

78. Jens, W. and Rehm, F. R. "Municipal Incineration and Air Pollution Control." Preprint. (Presented at the National Incinerator Conference, American Society of Mechanical Engineers, New York, May 20–24, 1966.)

79. Kaiser, E. R. "Refuse Composition and Flue Gas Analyses from Municipal Incinerators." Proc. Natl. Incinerator Conf., American Society of Mechanical Engineers, 1964. pp. 35–51.

80. "Atmospheric Emissions from Sulfuric Acid Manufacturing Processes. A Cooperative Study Project of the Manufacturing Chemists' Association, Inc." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, PHS-Pub-999-AP-13, 1965.

81. Doherty, R. E. "Current Status and Future Prospects, Cement Mill Air Pollution Control." Proc. Natl. Conf. Air Pollution, Washington, D.C., 1966, pp. 242-249.

82. "Motor Vehicles, Air Pollution, and Health. A report of the Surgeon General to the U.S. Congress." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Washington, D.C. 1962.

83. Mueller, P. K., Helwig, H. L., Alcocer, A. E., Gong, W. K., and Jones E. E. "Concentration of Fine Particles and Lead in Car Exhaust." American Society for Testing and Materials, Spec. Tech. Pub. 352, 1964, pp. 60–73.

84. Lee, R. E., Jr., Patterson, R. K., Crider, W. L., and Wagman, J. "Concentration and Particle Size Distribution of Particulate Emissions in Auto Exhaust." 1968. (Manuscript in Preparation).

85. Bowden, A. T., Draper, P., and Rowling, H. "The Problem of Fuel Oil Deposition in Open Cycle Gas Turbines." Am. Inst. Mech. Engrs. Proc., Vol. 167, pp. 291–300, 1953.

86. MacPhee, R. D., Taylor, J. R., and Chaney, A. L. "Some Data on Particles from Fuel Oil Burn-

ing." Air Pollution Control District, Air Analysis Division, Los Angeles, California, Analysis Paper 7, November 18, 1957.

87. Smith, W. S. "Atmospheric Emissions from Fuel Oil Combustion." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, PHS-Pub-999-AP-2, November 1962.

88. Smith, W. S. and Gruber, C. W. "Atmospheric Emissions from Coal Combustion. An Inventory Guide." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, PHS-Pub-999-AP-24, April 1966.

Chapter 2

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON SOLAR RADIATION AND CLIMATE NEAR THE GROUND

Table of Contents

Page

A. INTRODUCTION 35

B. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON VISIBLE RADIATION 35

C. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON TOTAL SOLAR RADIATION 38
  1. Physical Factors 38
  2. Seasonal Variations 39
  3. Weekly Variations 39
  4. Other Variations 40

D. INFLUENCE ON PRECIPITATION 40

E. RELATION TO WORLDWIDE CLIMATE CHANGE 42

F. SUMMARY 43

G. REFERENCES 44

List of Figures

Figure

2–1 Relation of Solar Transmissivity to Height Above Ground in "Polluted" and "Clean" Areas 36

2–2 Annual Variation of the Ratio of Illumination Levels in Central London and at Kew (•), and of Concentration of Particulate matter at kew (o) 37

2–3 Causes of Cyclical Diurnal Smoke Variations 41

2–4 Yearly Cycle (Averaged Over Six Years) of Deposited Pollutants 41

2–5 Precipitation Values at Selected Indiana Stations and Smoke-Haze Days at Chicago 42

List of Tables

Table

2–1 Approximate Association Between Atmospheric Aerosol Concentrations and Relative Solar Radiation Levels 39

2–2 Loss of Sun's Radiation in Three European Cities Over That in the Adjacent Country 40

2–3 Mean Number of Condensation Nuclei for Various Ranges of Dust Concentrations in City Air 40

Chapter 2

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON SOLAR RADIATION AND CLIMATE NEAR THE GROUND

A. INTRODUCTION

Particles in the atmosphere play several roles in the behavior and determination of the weather. Among the most obvious is the effect they have on the radiation from the sun. They scatter the light to greater or lesser extents in different wavelength regions, depending on their size, character, and concentration, and thus provide the sky with its variable hues, its colorful dawns and sunsets, and also dense hazes and dark urban palls. More subtle changes are occasioned by particles when they reduce the amount of solar energy that reaches the ground.

Particles play a less advertised but very essential role in the formation of clouds. Without them, liquid water clouds could not form except at supersaturations of several hundred percent. However, the sort of particles required (called condensation nuclei) are provided in overabundance by natural processes. Only to the extent that higher-than-normal nuclei concentrations can affect the cloud-forming process, does man's introduction of additional particulate material into the atmosphere produce changes in observed cloud structure and occurrence.

The kinds of particles which cause precipitation from clouds, in contrast to those necessary for cloud formation, are frequently in short supply in the atmosphere. In warm (above freezing) clouds, the requirement is a wide distribution of condensation nuclei, some of which are giant hygroscopic nuclei larger than 1μ. These "giants" promote the rapid growth of cloud droplets by producing some droplets large enough to fall with respect to the others. These grow rapidly by sweeping up smaller drops and very soon become massive enough to fall from the base of the cloud as rain.

Another kind of particle is required to stimulate the rapid transformation of cloud droplets into precipitation in supercooled (subfreezing) parts of clouds. Such particles are termed freezing nuclei. Without these, the water droplets would not freeze except at temperatures below −40°C. Once frozen, small ice particles grow rapidly at the expense of the surrounding water droplets and begin to fall as snow. They may later melt to become rain. Dramatic changes in cloud structure have been achieved by seeding supersaturated clouds with appropriate freezing nuclei. There is evidence, discussed below, that some observed changes in precipitation patterns in a few sections of the country can be ascribed to the inadvertent seeding of clouds by industrial contaminants.

Finally, tentative as our current information and understanding may be, the long-range potential effect of adding more and more particles to the atmosphere cannot be ignored. As more is learned about the general circulation of the atmosphere and the delicate balance between incoming and outgoing radiation (the "throttle on the atmospheric engine"), it seems increasingly possible that small changes such as those occasioned by increasing particle loads in the atmosphere may produce very long-term meteorological effects.

B. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON VISIBLE RADIATION

Stable particles of negligible fall velocity are probably the most common and persistent air pollutants. Their optical effects in produc-

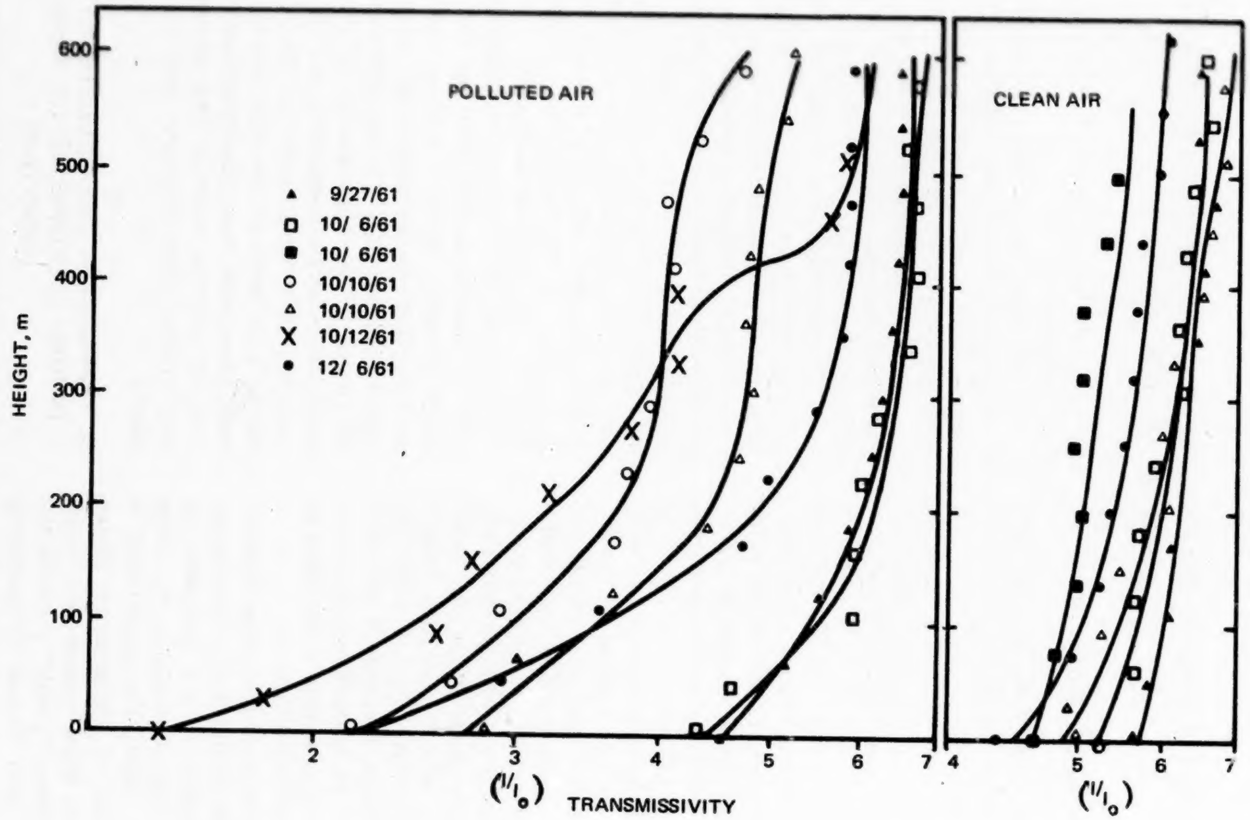

FIGURE 2–1. Relation of Solar Transmissivity to Height Above Ground in "Polluted" and "Clean" Areas. (This figure illustrates the effect of pollution on the attenuation of solar radiation reaching the ground.)

ing haziness, atmospheric turbidity, and a reduction of visibility which hampers the safe operation of aircraft and motor vehicles, are well known. (See Chapter 3.) The solar radiation transmissivity ($I[\lambda]/Io[\lambda]$ where $I[\lambda]$ is the intensity of the normal-incidence solar radiation and $Io[\lambda]$ is the extra-terrestrial value at this wavelength) varies with height and is strongly influenced by the dust loading in the lower atmosphere.[1] Figure 2–1 illustrates the relation of transmissivity at $\lambda = 5000$ Å to height in the atmosphere above an urban area under varying conditions of particulate loading. In the case of heavily polluted air, this radiation may be reduced by more than one-half in the lowest 300 meters of the atmosphere. There is also an increased attenuation of visible radiation near the ground in clean air but the amount is small by comparison (Figure 2–1).

It is well known that the reduction or attenuation of visible radiation in industrialized urban areas, and the attendant gloom caused by excessive concentrations of suspended particles in the air, create a need for additional artificial illumination in offices, factories, and homes, and produces added economic stresses. This is particularly true in winter, when the days are shorter and the particulate content of the urban air is greater because of greater combustion of fuels. Data given by Shepherd [1] illustrate some relationship, shown in Figure 2–2, between particle concentrations and relative visible radiation levels in London during summer and winter months. Daylight illumination was measured at two sites, one in central London, the other in Kew Observatory, a slightly less heavily polluted area 13 miles WSW of the first site. As another example, the average loss of sunlight in the city of Leningrad compared to the countryside was estimated to be 40 percent over the period of a year; in winter the loss was estimated to reach 70 percent in the city, while in summer the loss was about 10 percent.[3]

Haze in the atmosphere due to forest fires, dust storms, or smoke from other sources, may become so concentrated at times that the sun appears red in the sky despite the absence of clouds. Just after sunrise or just before sunset, the haze may reduce the intensity of the direct sunlight so much that one can look directly at the sun without eye strain or injury. In other words, haze in the atmosphere due to suspended particles scatters the light from the sun, making it appear dim.

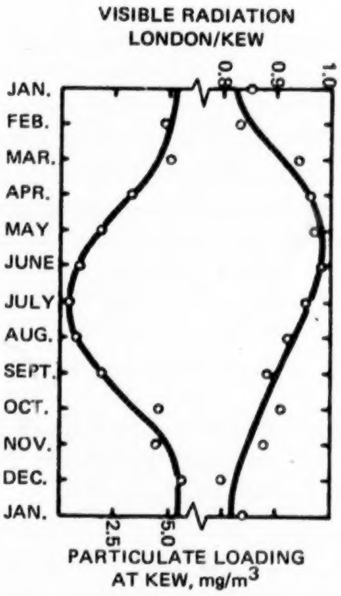

FIGURE 2–2. Annual Variation of the Ratio of Visible Radiation Levels in Central London and at Kew (●), and of Concentration of Particulate Matter at Kew (o) [1] (This figure indicates the relative attenuation of light at two sites in a large city.)

Scattering of the sunlight by the particles makes the air seem "turbid," and an optical device called the Volz sun photometer has been devised to measure this "turbidity" quantitatively.[4] Starting with equations 3–2 in Chapter 3 (Lambert's Law), one calculates from the readings of this instrument a "turbidity coefficient," B, related to the extinction coefficient b in equation 3–2, Chapter 3, or more specifically to b_{scat} defined there. The relationship between B and b is outlined by McCormick and Baulch,[2] who found that in a city B_z, the value of B measured by the Volz instrument at a height of z meters above the ground, is related to B_o, the value of B measured near ground level, by the equation

$$B_z = B_o e^{-0.00346z} \qquad (2\text{–}1)$$

From scattering theory, McCormick and Baulch estimate that at any height, z, up to 200 meters above the ground, the number of particles per cubic meter, n′ (z), in the radius range between 0.7μ and 1μ is given by

$$n'(z) = 17.3 \times 10^9 B_o e^{-0.00346z} \quad . \qquad (2\text{–}2)$$

Both of these equations are rough approximations for light-wind, clear-sky conditions in a city during moderate to heavy pollution situations. In terms of mass loading near the ground, [m′ (o)],

$$m'(o) \approx 10^3 B_o \ (\mu g m^{-3}) \qquad (2\text{–}3)$$

C. EFFECTS OF PARTICULATE MATTER IN THE ATMOSPHERE ON TOTAL SOLAR RADIATION

Landsberg[5] reports that cities in general receive 15 percent to 20 percent less insolation (on a horizontal surface) than do their rural environs. Insolation, as used here, means the total solar radiation received at the earth's surface per unit area per unit time. This discussion considers the part that airborne particles take in the diminution of insolation received by cities.

1. Physical Factors

The attenuation of solar radiation through the atmosphere is caused by a number of physical factors:[2, 6–10]

1. Scattering of radiation, known as Rayleigh scattering, by the air molecules, such as N_2 and O_2, and particles in size ranges less than the wavelength of the solar radiation (the scattering coefficient is inversely proportional to the fourth power of the wavelength of the incident radiation; hence the short wavelength radiation is scattered most, so that the sky appears to be blue);
2. Selective absorption by the gaseous constituents of the atmosphere such as ozone and CO_2, and by water vapor; and
3. Scattering and absorption by atmospheric dusts and particulate matter of size greater than in (a).

The attenuation of solar radiation by water vapor and ozone is negligible in the visible wavelength region usually studied.[6, 7, 11]

The scattering of light by aerosol particles in ambient air is a complicated process. Part of the incident light is transmitted, part is reflected in all directions either at the front surface of the particle or at an internal discontinuity, and part is absorbed. The transmission factor for scattering is a function of the wavelength of the incident light and the physical qualities of the scattering medium. In simple cases where the form, size, and composition of the scattering particles are known, the factor can be derived on a theoretical basis and is known as "Mie" scattering. Chapter 3 provides a more extended discussion.

Diffuse radiation is an important factor in the amount of heat and light received at any given location.[3, 10] The intensity and spectral distribution of direct sunlight and scattered daylight, and the varation of intensity with time of day, season, latitude, altitude, and atmospheric conditions such as turbidity, are important because they affect photosynthesis in plants and the distribution of plants and animals on earth, the weathering of natural and man-made materials, climate, and illumination for human activity.[9] The percentage of direct solar radiation which will remain after its attenuation by smoke and other atmospheric constituents depends both on the atmospheric turbidity caused by the smoke, and on the altitude of the sun above the horizon, as well as on the other factors[3] previously noted.

Except in cases of heavy particulate pollution of the atmosphere, such as may occur in large urban centers or heavy industry areas, it appears that the effect of turbidity is to scatter radiation out of the direct solar beam and to add an almost equal amount of radiation to the diffuse beam arriving from the rest of the sky by forward-scattering. In cases of heavy particle concentrations, however, the loss from the direct solar beam greatly exceeds the gain in the downward scattered beam, the difference being lost to back-scattering off the top of the pollution layer and to absorption within the polluted layer or column.

Studies indicate a fair approximation of the association between atmospheric aerosol concentration and relative solar radiation levels, as shown in Table 2–1.

Table 2–1.—APPROXIMATE ASSOCIATION BETWEEN ATMOSPHERIC AEROSOL CONCENTRATIONS AND RELATIVE SOLAR RADIATION LEVELS.

Aerosol concentration, $\mu g\ m^{-3}$	Solar radiation, percent of value for 100 $\mu g\ m^{-3}$	
	Total	Ultraviolet
50	105	104
100	100	100
200	95	92
400	90	77

The reduction in ultraviolet light reaching the surface may be as important as the attenuation of other, longer wave, components of solar radiation. Available data are sparse but one study [12] indicates that ultraviolet intensity decreased by about 7.5 percent for each 100 $\mu g/m^{-3}$ increase in aerosol content of the atmosphere with an average deficiency of over 20 percent in the city as compared with its environs in winter. Some studies suggest that a 5 percent reduction in total solar radiation resulting from smoke almost completely eliminates the ultraviolet component.[3, 12] Even in a comparatively clean atmosphere, the effective ultraviolet drops to very low values when the sun's elevation is below 30 degrees.[13]

The net influence of atmospheric turbidity on surface temperature is uncertain, but for typical turbidity indices in the United States, it is likely to be small. The effect on solar radiation (warming in the upper region of the pollution layer due to extinction by the upper region) tends to be compensated by the effect on radiation returning to space from the earth's surface and atmosphere. The extent to which the solar radiation effect prevails over the terrestrial radiation effect, or vice versa, is dependent on a number of factors which include:

1. the time of day and year (on which the intensity of solar radiation is itself dependent);
2. the total mass and vertical distribution of the particles;
3. the size distribution of the particles;
4. relative humidity, which, in the case of hygroscopic aerosols, may alter the effective absorptivity and reflectivity of the particles; and
5. temperature of the air and ground (on which the intensity of the terrestrial radiation depends).

2. Seasonal Variations

The concentration of suspended particulate matter which ranges from less than 60 $\mu g/m^3$ to 1700 $\mu g/m^3$ in various American cities shows a notable annual variation. Autumn and winter particulate levels are invariably highest, and summer levels lowest. The weakening of radiation caused by smoke is less during the summer when the sun is high than in the winter. This would be true even if there were the same degree of pollution in the air. The losses in intensity of direct total solar radiation during its passage through an atmosphere polluted by smoke may become as high as one-third in the summer and two-thirds in the winter.[3]

Landsberg [5] has summarized the radiation loss data compiled by Steinhauser [13] for three Central European cities—Frankfurt, Leipzig, Vienna—and their adjacent rural areas, for the four seasons of the year. There are contrasts in loss of solar radiation between spring or summer and winter, as will be seen in Table 2–2. Steinhauser and coworkers [13] also reported that Vienna receives a lesser total radiation than the nearby countryside: in winter 85 percent of the total solar radiation of suburban Hoche Warte, in spring 92 percent, in summer 92 percent, and in autumn 87 percent.[8, 13] The absorption is strongest in the short (ultraviolet) wavelengths.[8, 11, 13, 14]

This annual cycle is observed to be a function of latitude, dependent on the changing midday sun angle. The scattered radiation during the summer months (May-August) may amount to approximately 60 percent to 65 percent of the direct radiation at 60° latitude and to about 45 percent at 40° latitude.

3. Weekly Variations

In addition to the seasonal and diurnal variations or patterns of total solar radiation in urban communities, a weekly cycle of intensity of total solar radiation has been observed,[15–17] which is related to the weekly cycle of industrial and commercial activity. In general, the total solar radiation received is inversely related to the concentration of smoke and suspended particles; thus solar radiation measurements may be used, in the absence of clouds, as a crude index of particulate air pollution.

Table 2-2.—LOSS OF SUN'S RADIATION IN THREE EUROPEAN CITIES OVER THAT IN THE ADJACENT COUNTRY.[5]

Season	Solar Elevation			
	10°	20°	30°	45°
	Percent	*Percent*	*Percent*	*Percent*
Winter	36	26	21	—
Spring	29	20	15	11
Summer	29	21	18	14
Autumn	34	23	19	16

Mateer[17] studied the total solar and sky radiation patterns in metropolitan Toronto from October 1937 to 1960 and compared the radiation received on Sundays to the average of the weekday radiation readings at the same central site. The average radiation on Sundays for this period was 313.8 langleys (1 langley$=$1 g cal/cm^2) while the mean for the weekday radiation was 305.2 langleys, a difference of 8.6 langleys or 2.8 percent. The probability of obtaining such a difference by chance was less than 0.5 percent. Thus, while a real difference in radiation exists between Sundays and weekdays, the magnitude is rather small.

4. Other Variations

Hand[18] has noted that the average daily solar radiation in various cities was significantly higher over the entire year 1932 than over the prior year. The largest increase in solar radiation was found to be in New York City (+21.9 percent), while Pittsburgh and Washington showed yearly increases of 6.2 percent and 8 percent respectively. The business depression was probably a major factor in this increase. Records for New York, Chicago, and Pittsburgh for the year 1932 showed the marked diminution in dust and smoke levels resulting from the falling off in the amount of manufacturing during this period.

D. INFLUENCE ON PRECIPITATION

There is evidence that some of the particles introduced into the atmosphere by man's activities can act as nuclei in processes which affect the formation of clouds and precipitation.

Condensation nuclei in the size range greater than 1μ are often made up of hygroscopic particles.[19, 20] Because of their affinity for water, these particles play an important role in the transformation and condensation of water vapor into liquid water droplets or solid ice particles, and are of vital importance in the formation of fog, clouds, and rain. Combustion products of man's industry and technology, as well as volcanic eruptions and ocean spray, are significant sources of these nuclei. A parallelism exists between the concentration of dust and that of condensation nuclei in city air. (See Table 2–3.)

Table 2-3.—MEAN NUMBER OF CONDENSATION NUCLEI FOR VARIOUS RANGES OF DUST CONCENTRATIONS IN CITY AIR.[19]

Number of dust particles per cm^3	Mean number of condensation nuclei per cm^3
<500	189,000
500–999	211,000
>999	223,000

A pronounced parallelism can also be found with respect to diurnal and annual variations in the contents of atmospheric dust and nuclei.[16, 19–22] The excess production of condensation nuclei in the air over cities is a long established fact,[5, 20, 23] and has been reaffirmed with many amplifying circumstances.[22, 23] Evidence has been presented that giant nuclei, which may initiate the coalescence process, are more abundant in industrial areas than elsewhere.[16]

The diurnal, weekly, and yearly cycles of both suspended and dustfall particle concentrations, correspond closely to man's pattern of activities and combustion requirements. There are usually two diurnal peaks (see

R

Figure 2–3), greater midweek concentrations compared to Sundays, and greater mean concentrations in winter than in summer (see Figure 2–4).[16]

A correlation has been found between patterns of precipitation over cities and the adjacent countryside and the variations of particle concentrations in the atmosphere.[22–24] The influence of cities on precipitation is complex; however, there is a general tendency for urban factors to increase precipitation.[23] These factors, not necessarily in order of importance, are:

1. water vapor addition from combustion sources and processes;
2. thermal updrafts from local heating;
3. updrafts from increased friction turbulence;
4. added condensation nuclei leading to more ready cloud formation; and,
5. added nuclei which may act as freezing nuclei for super-cooled cloud particles.

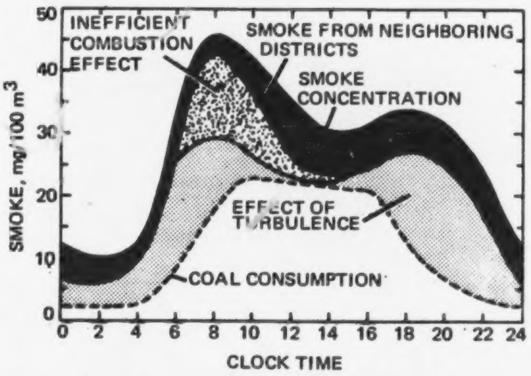

Figure 2–3. Causes of Cyclical Diurnal Smoke Variations.[18] (This figure shows causes of changes in the diurnal airborne particulate concentration in an urban location.)

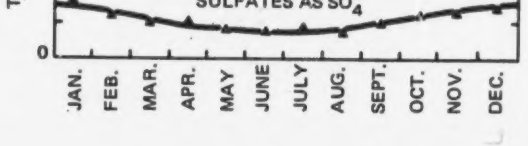

Figure 2–4. Yearly Cycle (Averaged Over Six Years) of Deposited Pollutants. (This figure illustrates the variation of amount of pollutants deposited at Leicester town hall averaged over the six years ending March 1939, and shows the greater mean concentration in winter as compared with summer.)

Landsberg [23] cites as evidence the gradual increase of rainfall which followed the growth of Tulsa, Oklahoma, from a village to a city in five decades and the concomitant increase in particle concentrations. A study by Kline and Brier [25] in metropolitan Washington, D.C., indicates that there is a considerably higher level of freezing nuclei in the metropolitan area than there is in the adjacent countryside.

Ashworth [24] first suggested the correlation between the weekly cycle of smoke in industrial areas and that of precipitation. Frederick,[26] in a more recent analysis, showed a definite minimum Sunday rainfall for a ten-year period in Louisville, Pittsburgh, and Buffalo.[23, 26] Precipitation occurred in these less often on Sundays than on other days of the week, and the average rainfall was less for Sundays than for weekdays. A strong city influence is also suggested in the snow patterns in Toronto.[27]

An interesting and significant increase in precipitation has been observed at La Porte, Indiana, since 1925. La Porte is 30 miles east of the large complex of heavy industries in the metropolitan Chicago area. Changnon [28] compared precipitation at La Porte, Valparaiso, and South Bend, Indiana, with a five-year moving average of the number of smoky and hazy days in Chicago (Figure 2–5). The temporal distribution of the smoke-haze days after 1930 is rather similar to the La Porte curve. A notable increase in smoke-haze days began in 1935, becoming more marked after 1940, coincident with the sharp increase in the La Porte precipitation curve. The reduction in the frequency of smoke-haze days after the peak reached in 1947 also generally matches the decline of the La Porte curve since 1947.[28]

Stout [29] has shown that the shape of the time-series curve for La Porte precipitation also generally matched a time-series curve for annual steel production in the Chicago industrial complex. Between 1905 and 1965, peaks in steel production, which occurred when production in most other industries was also high, were all associated with high points in the La Porte precipitation curve.

The effect of industrial pollution on precipitation has also been studied by Telford.[30] He found that smoke from steel furnaces was a prolific source of freezing nuclei, increasing counts by a factor of 50 over those in nearby clean air. He concluded that there should be increased rainfall downwind of such installations.

E. RELATION TO WORLDWIDE CLIMATIC CHANGE

Theoretical considerations and empirical evidence indicate that atmospheric turbidity, itself a function of aerosol concentration, is an important factor in the heat balance of the earth-atmosphere system. The observed increase in turbidity over the past few decades may play a role in the reported decrease in worldwide air temperature since 1940 by increasing the planetary albedo.[7, 31]

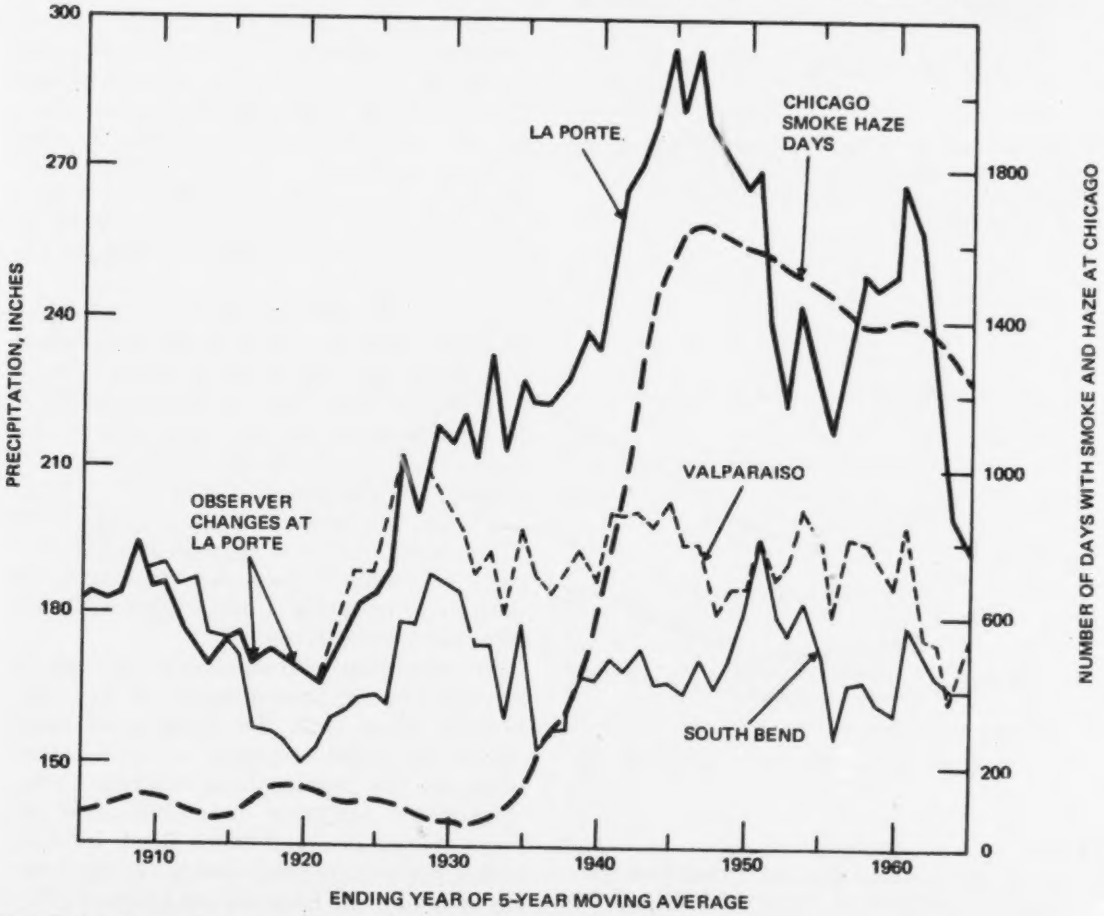

FIGURE 2–5. Precipitation Values at Selected Indiana Stations and Smoke-Haze Days at Chicago. (This figure shows the way in which precipitation trends at La Porte follow the haze changes in Chicago. The results are plotted as five-year moving averages.)

Ångstrom estimated roughly that a change in the albedo from 0.40 to 0.41 corresponds to a change in the mean temperature of the earth-atmospheric system of close to 1° C.[32] Humphreys [33] made similar calculations with roughly the same results, and also showed that the interception of outgoing radiation by fine atmospheric dusts is wholly negligible in comparison with the interception of incoming solar radiation. Temporal and spatial changes in the atmospheric turbidity of 100 percent, corresponding to albedo changes of 10 percent to 15 percent, from one day to the next or from one locality to another, are very commonplace.[31] Even though these figures may well overestimate the actual changes brought about in atmospheric temperatures, the course of atmospheric turbidity over the earth is an important climatic factor.

There are data available [31, 34] from which the trend in turbidity during the past century can be estimated. Ångstrom gave 0.098 as the value of the mean annual turbidity at Washington, D.C. (1903 to 1907),[11] and 0.024 as the value at the Davos Observatory, Switzerland (1914 to 1926). The values for Washington were determined from data on solar transmission (by wavelength) published by the Smithsonian Institution; those for Davos were from data attributed to Lindholm on dust absorption. In 1962, determinations of the atmospheric turbidity were begun at the Continuous Air Monitoring Program station [31, 35] of the Public Health Service, near the Smithsonian Institution; the mean annual turbidity recorded for 1962 to 1966 was 0.154,[35] a 57 percent increase over the 1903 to 1907 values. From 1957 to 1959, determinations of atmospheric turbidity were again made for Davos by Valko [36] and were given as 0.043, a 70 percent increase.

When the scattering theory with a Junge distribution of particle size [4] is used, the values of turbidity change imply an increase in the average annual number of aerosol particles, in the range of 0.1μ to 1μ radius, of 2.8×10^7cm^2 and 0.95×10^7/cm^2 over Washington and Davos respectively, during the periods shown. Nearly two-thirds of the Washington increase might be attributed to the increased population and urbanization of the District since the turn of the century. A significant remainder, however, as judged by the Davos increase, may be indicative of a much more general buildup of atmospheric aerosol.

When the above facts are put together, they indicate that for Washington, D.C., during the period 1903 to 1966, there has been a possible decrease of nearly 3 percent in the total available solar energy at ground level and a possible increase in the average annual number of aerosol particles of 2.8×10^7cm^2. The net effect of this apparent secular increase in turbidity (which from the Davos, Switzerland, and other evidence [34] appears likely to be worldwide) is probably to increase the mean albedo of the planet and reduce the mean temperature of the earth-atmosphere system.

The increase in atmospheric turbidity consequent upon volcanic eruptions may have temporary effects on atmospheric temperatures. Mitchell [37] concluded that temperatures over large areas of the world may be depressed by 0.5° F or more in the first or second year following an unusually violent eruption. However, McCormick and Ludwig [31] suggest that the effects of man's pollution of his environment are increasing steadily along with the world population. The emission of long-lived particles, keeping pace with the accelerated worldwide production of CO_2, may well be leading to the decrease in world air temperature in spite of the apparent buildup of CO_2.[38]

F. SUMMARY

Atmospheric particles scatter and absorb light from the sun, thus reducing the visible radiation available to cities and the solar radiation that reaches the earth. The gloom due to reduced illumination in urban areas creates a need for artificial lighting in offices, factories, and homes and produces related economic stresses. The average year-round illumination (i.e., the portion of the spectrum that is visible to the eye) may be reduced by one-third or more in some cities. Daylight illumination in the center of London, for example, was found to be 20 percent less than that found at a slightly less-polluted part of the city quite near the center.

Part of the sunlight reaching the earth comes from the direct beam and part from that scattered from the rest of the sky. The *total* solar energy reaching the earth is the sum of both the direct and the scattered radiation. In general, cities receive 15 percent to 20 percent less total solar radiation than do rural environments, although the net reduction may be considerably greater under some circumstances. For a typical urban area in the United States, with a geometric mean annual concentration of roughly 100 $\mu g/m^3$, the total sunlight is reduced approximately 5 percent for every doubling of particle concentration. This effect is more pronounced on the ultraviolet portions of the spectrum.

Diurnal, weekly, and yearly cycles of both suspended particulates and dustfall particles correspond closely to man's pattern of activities and his combustion requirements. There are usually two daily peaks, greater midweek concentrations compared with Sundays, and greater mean concentrations in autumn and winter than in spring and summer. The seasonal variations are due largely to the use of coal and heavy fuel oil for heating purposes. Solar radiation attenuation patterns also show weekly and seasonal variations. On weekdays, the attenuation is slightly more than on Sundays, and the losses in intensity of the direct beam during its passage through an atmosphere polluted by smoke may become as high as one-third in the summer and two-thirds in the winter. Even variations from year to year have been noted; for example, levels of total solar radiation measured in various cities were significantly higher in 1932 than in the prior year, undoubtedly because of the decline in manufacturing and industrial activity brought about by the depression. Reductions in the intensity of solar radiation or changes in its spectral distribution have significance for the photosynthesis of vegetation, the distribution of plants and animals on the earth, the weathering of natural and manmade materials, and man's aesthetic enjoyment and physical well-being.

The increased emission of fine particles into the atmosphere also may cause changes in the delicate heat balance of the earth-atmosphere system, thus altering worldwide climatic conditions. The rise in atmospheric turbidity increases the planetary reflectivity, or albedo, and reduces the solar energy available to maintain surface temperatures. This phenomenon may be responsible for the reported decrease in worldwide temperature since the 1940's. Comparisons at different sites over the world covering periods as long as fifty years suggest that a general worldwide rise in turbidity may be taking place. This may well be indicative of a gradual buildup of worldwide background levels of suspended particulates. The increasing levels of atmospheric carbon dioxide resulting from man's combustion of fuels probably exerts an opposite effect on worldwide temperatures, but the emission of long-lived particles to the air may gradually depress world air temperature despite the apparent buildup of carbon dioxide.

Some of the particles introduced into the atmosphere by man's activities also can affect the weather by serving as condensation nuclei that influence the formation of clouds, rain, and snow. The large airborne particles generated in metropolitan areas serve as a base for the condensation of moisture and lead to the rapid formation of rain droplets or ice crystals. Patterns of precipitation over cities and the adjacent countryside have shown a correlation with particle concentrations in the atmosphere. Some cities display a definite minimum rainfall on Sundays, when particulate levels are usually lowest, and records over several decades reveal that rainfall levels may increase with the concomitant rise in particulate levels that generally accompanies urban growth. In addition, long-term changes in the frequency of smoke-haze days in one city may affect rainfall levels in a nearby downwind city.

Thus airborne particles can, through a number of mechanisms, influence man's surroundings and have considerable impact on weather and climatic conditions.

G. REFERENCES

1. Sheppard, P. A. "The Effect of Pollution on Radiation in the Atmosphere." Intern. J. Air Pollution, Vol. 1, pp. 31–43, 1958.

2. McCormick, R. A. and Baulch, D. M. "The Variation with Height of the Dust Loading over a City as Determined from the Atmospheric Turbidity." J. Air Pollution Control Assoc., Vol. 12, pp. 492–496, 1962.
3. Sheleikhovskii, G. V. "Smoke Pollution of Towns." Akademiya Kommunal'nogo Khzyaistva im. K. D. Pamfilova. [Academy of Municipal Economy im. K. D. Pamfilova.] Izdatel'stvo Ministerstva Kommunal'nogo Khozyaistva RSFSR, Moskva-Leningrad. 1949. (Translated from Russian and published for the National Science Foundation, Washington, D.C. by the Israel Program for Scientific translations, Jerusalem, 1961.)
4. Volz, F. "Photometer mit Selen-Photoelement zur spektralen Messung der Sonnenstrahlung und zur Bestimmung der Wellenlangenabhangigkeit der Dunsttrubung." Arch. Meterol. Geophys. Bioklimatol., Ser. B, Vol. 10, pp 100–131, 1959.
5. Landsberg, H. "Physical Climatology." 2nd edition, Gray, DuBois, Pennsylvania, 1958, pp. 317–326.
6. Ångstrom, A. K. "On the Atmospheric Transmission of Sun Radiation II." Geograph. Ann. (Stockholm), Vol. 12, pp. 139–159, 1930.
7. McCormick, R. A. "Atmospheric Turbidity." Preprint. (Presented at the 60th Annual Meeting, Air Pollution Control Association, Cleveland, Ohio, June 11–16, 1967.)
8. Stagg, J. M. "Solar Radiation of Kew Observatory." Air Ministry, Meteorological Office, London, Geophysical Memoirs 86, 1950.
9. Gates, D. M. "Spectral Distribution of Solar Radiation at the Earth's Surface." Science, Vol. 151, pp. 523–529, 1966.
10. Robinson, N. "Solar Radiation." Elsevier, Amsterdam, London and New York, 1966.
11. Ångstrom, A. K. "On the Atmospheric Transmission of Sun Radiation and Dust in the Air." Geograph. Ann. (Stockholm), Vol. 11, pp. 156–166, 1929.
12. Shrader, J. H., Coblentz, M. H., and Korff, F. A. "Effect of Atmospheric Pollution upon Incidence of Solar Ultraviolet Light." Am. J. Public Health, Vol. 19, pp. 717–724, 1929.
13. Steinhauser, F., Eckel, O., and Sauberer, F. "Klima und Bioklima von Wien." Wetter und Leben, Vol. 7, 1955, 120 pp.
14. Kenrick, G. W. and Ortiz, H. "Measurements of Ultra-Violet Solar Radiation in Puerto Rico." Trans. Am. Geophy. Union, 19th, 1938, pp. 138–140.
15. Meetham, A. R. "Atmospheric Pollution in Leicester. A Scientific Survey." Dept. of Scientific and Industrial Research, London, Atmospheric Pollution Research Technical Paper 1, 1945.
16. Meetham, A. R. "Atmospheric Pollution: Its Origin and Prevention." Pergamon Press, New York, 1961.
17. Mateer, C. L. "Note on the Effect of the Weekly Cycle of Air Pollution on Solar Radiation at Toronto." Intern. J. Air Water Pollution, Vol. 4, pp. 52–54, 1961.
18. Hand, I. F. "Solar Radiation Measurements During December 1932." Monthly Weather Rev., Vol. 60, pp. 256–257, 1932.
19. Neuberger, H. "Condensation Nuclei: Their Significance in Atmospheric Pollution." Mech. Eng., Vol. 70, pp. 221–225, 1948.
20. Landsberg, H. "Atmospheric Condensation Nuclei." Erg. Kosm. Physik., Vol. 3, pp. 155–252, 1938.
21. Giner, R. and Hess, V. F. "Studie uber die Verteilung der Aerosole in der Luft von Innsbruk und Umgebung." Gerlands Beitr. Geophys., Vol. 50, pp. 22–43, 1937.
22. Georgii, H. W. "Probleme und Stand der Erforschung des Atmospharischen Aerosols." Ber. Deutsche Wetterdienste., Vol. 7, pp. 44–52, 1959.
23. Landsberg, H. "City Air—Better or Worse." In: Air Over Cities Symposium, U.S. Dept. of Health, Education, and Welfare, Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio, Technical Report A62–5, 1962, pp. 1–22.
24. Ashworth, J. R. "The Influence of Smoke and Hot Gases from Factory Chimneys on Rainfall." Quart. J. Roy. Meteorol. Soc., Vol. 55, pp. 341–350, 1929.
25. Kline, D. B. and Brier, G. W. "Some Experiences on the Measurement of Natural Ice Nuclei." Monthly Weather Rev., Vol. 89, pp. 263–272, 1961.
26. Frederick, R. H. Personal communication. U.S. Weather Bureau.
27. Potter, J. G. "Changes in Seasonal Snowfall in Cities." Canadian Geographer, Vol. 5, pp. 37–42, 1961.
28. Changnon, S. A. Jr. "The La Porte Weather Anomaly, Fact or Fiction." Bull. Am. Meteorol. Soc., Vol. 49, pp. 4–11, 1968.
29. Stout, G. E. "Some Observations of Cloud Initiation in Industrial Areas." In: Air Over Cities Symposium, U.S. Dept. of Health, Education, and Welfare, Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio, Technical Report A62–5, 1962, pp. 147–153.
30. Telford, J. W. "Freezing Nuclei from Industrial Processes." J. Meteorol., Vol. 17, pp. 676–679, 1960.
31. McCormick, R. A. and Ludwig, J. H. "Climate Modification by Atmospheric Aerosols." Science, Vol. 156, pp. 1358–1359, 1967.
32. Ångstrom, A. K. "Atmospheric Turbidity, Global Illuminators and Planetary Albedo of the Earth." Tellus, Vol. 14, pp. 435–450, 1962.
33. Humphreys, W. J. "Volcanic Dust and Other Factors in the Production of Climatic Changes and their Possible Relation to Ice Ages." Bull. Mount. Weather Observatory, Vol. 6, pp. 1–34, 1914.

34. Peterson, J. T. and Bryson, R. A. "Atmospheric Aerosols: Increased Concentrations During the Last Decade." Science, Vol. 162, pp. 120–121, 1968.

35. "Atmospheric Turbidity Report." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Cincinnati, Ohio. (Quarterly)

36. Valko, P. "Untersuchung über die vertikale Trübungsschichtung der Atmosphäre." Arch. Meteorol. Geophys. Bioklimatol., Ser. B, Vol. 11, pp. 143–210, 1961.

37. Mitchell, J. M., Jr. "Recent Secular Changes in Global Temperatures." Ann. N.Y. Acad. Sci., Vol. 95, pp. 235–250, 1961.

38. "Restoring the Quality of Our Environment." Report of the Environmental Pollution Panel, President's Science Advisory Committee, The White House, Washington, D.C., November 1965, pp. 111–131.

Chapter 3

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VISIBILITY

Table of Contents

Page

A. INTRODUCTION 51

B. IMPORTANCE OF ATMOSPHERIC AEROSOLS TO THE GENERAL OPTICAL PROBLEM 51

C. PHYSICAL RELATIONSHIPS BETWEEN VISIBILITY AND PARTICLE CONCENTRATION 52

D. COMPLICATIONS AND LIMITATIONS IN THE VISIBILITY PROBLEM 53
  1. Smoke Plumes 53
  2. Natural Aerosols and Hazes 53
  3. Fogs 54

E. THE LOW-HUMIDITY, WELL-AGED HAZE 54
  1. Size Distribution 54
  2. Mie Solutions 54
  3. Dependence of Extinction of Particle Size for the Atmospheric Case
  4. The Mass-Light Scattering Relationship 55

F. DETERMINATION OF WELL-DEFINED CASES OF MASS-VISIBILITY RELATIONSHIPS 58

G. METHODS FOR DETERMINING LIGHT SCATTERING COEFFICIENT-MASS CONCENTRATION RELATIONSHIP 59

H. SUMMARY 60

I. REFERENCES 61

List of Figures

Figure

3–1 Four Typical Measured Size Distributions of Atmospheric Suspended Particles Together with the Corresponding Visibilities 54

3–2 The Relation of Scattering Cross-Section to Particle Size for Particles of Three Different Refractive Indices 55

3–3 Cross Section Curve for Typical Atmospheric Size Distribution 55

3–4 The Dependence of Scattering Coefficient (m^{-1}) on Volume of Aerosol Particles (μ^3/cm^3) Calculated from Measured Size Distributions 56

3–5 Measured Dependence of Mass of Aerosol Particles per Volume ($\mu g/m^3$) on the Light Scattering Coefficient (m^{-1}) in Seattle, November-December 1966 56

3–6 Histogram of Equivalent Visual Range-Mass Concentration Product at Several Locations 57

3–7 Relation Between Visual Range and the Mass Concentration 57

3–8 Three Horizontal Profiles through the City of Seattle Taken under Differing Meteorological Conditions 59

List of Tables

Table *Page*

3–1 Relation Between Equivalent Visual Range and Particle Concentration 57

3–2 The Relative Humidity at which Phase Change Occurs in Some Deliquescent Aerosols 59

49/50

Chapter 3

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VISIBILITY

A. INTRODUCTION

One of the dramatic effects of air pollution is a degradation of the visibility. Visibility in the atmosphere is reduced by two optical effects which air molecules and aerosol particles have upon visible radiation. One is the attenuation by the molecules and particles of the light passing from object to observer. It is the result both of absorption of light and of the scattering of light out of the incident beam. The light received from the object and that received from its background are diminished by this attenuation, and the difference between the two (contrast) is consequently diminished with the result that the eye's ability to distinguish the object from its background is reduced. The other optical effect that degrades the contrast between object and background is the illumination of the intervening air which results when sunlight is scattered into the line of sight by the molecules and particles in the line of sight. It is a common observation that dark objects become progressively lighter in shade as they become more distant. The most distant mountain that one can distinguish typically is almost as light or bright as its sky background.

In those cases where a clear-cut relationship can be shown to exist between visibility and the mass of suspended particles per volume of air (mass concentration), it is possible to use some of the meteorological visibility records to infer the amount of pollution in past years as well as to study trends. In fact, many such studies have been made even without a substantive knowledge of the relationship. Holzworth[1] used existing visibility data both as an indication of the amount of pollution for comparison of an urban (Columbus, Ohio) area with a rural area, and for the qualitative inference of trends of both increasing and decreasing amounts of smoke or other atmospheric aerosol. Robinson[2] also discussed the use of meteorological visibility records and their interpretation. Both Holzworth[1] and Robinson[2] demonstrated that visibility degradation can be associated with air pollution. Neither, however, developed the sort of correlation between mass of suspended particulate matter per volume of air and visibility that is needed for air quality criteria. Robinson indicated doubt in the existence of a generally applicable relationship between mass concentration and visibility. This doubt was based on experimental results as well as on the complexity of the problem. Recent experimental and theoretical advances make possible some useful conclusions, and this chapter will define a simple relationship between mass of suspended particles and visibility, specify the circumstances under which it can be expected to be reliable, and describe the conditions which are too complex for this simple treatment.

In the United States, the words *visibility* and *visual range* are usually used synonymously to mean the distance at which it is just possible to perceive an object against the horizon sky. Middleton[3] reports that the originator of both terms intended that only visual range be considered as a distance, while visibility should convey a more qualitative judgment about the clearness of seeing. In this chapter, however, the terms visibility and visual range will be used interchangeably to mean a distance.

B. IMPORTANCE OF ATMOSPHERIC AEROSOLS TO THE GENERAL OPTICAL PROBLEM

Decreased visibility obviously interferes with certain important human activities, such as the safe operation of aircraft and automobiles and the enjoyment of scenic vistas. The effect of decreased visibility on the large-scale operations of commercial aircraft in metropolitan areas is a problem of growing concern. The Federal Air Regulations of 1967[4] (paragraphs 91.105 and 91.107) prescribe limitations on aircraft operation that become increasingly severe as visibility decreases below five miles. Most restrictions are invoked when the visibility is below three miles. In areas with a high density of aircraft traffic, visibilities much below five miles tend to slow down operations by maintaining larger separations between aircraft. Even though most commercial airplanes always fly under Instrument Flight Rules rather than Visual Flight Rules, good visibility increases both the safety and permitted traffic density. Light airplane operations are limited even more severely when visibility is less than three miles, because of their limited instrument flight capability.

A 1963 report by the Civil Aeronautics Board to the Committee on Public Works, U.S. Senate,[5] states that records of both automobile and aircraft accidents show cases where poor visibility due to smoke and air pollution was an important causal factor. Evidence presented at Federal air pollution abatement conferences[6, 7] shows the existence of air pollution that curtails visibility, endangering the safety of people traveling by both land and air, and in addition, causing inconvenience and economic loss to the public and to transportation companies due to disruption of traffic schedules.

C. PHYSICAL RELATIONSHIPS BETWEEN VISIBILITY AND PARTICLE CONCENTRATION

Many derivations of visibility theory have been published. Although Robinson's[2] approach is directed towards the air pollution problem, Middleton's[3] book presents a more complete view of the problem of atmospheric clarity.

For the simplest case of attenuation of a light beam along its path, the intensity, I, decreases by an increment dI over the increment of path dx. The relationship between intensity and distance is:

$$\frac{dI}{I} = -b\,dx \qquad (3\text{–}1)$$

or, in integrated form,

$$I = I_0 e^{-bx} \qquad (3\text{–}2)$$

where b is the extinction coefficient assumed to be constant over x, and I_0 represents the intensity of light at $x = 0$.

The extinction coefficient, b, is the sum of four terms:

1. the scattering coefficient of the air molecules, b_{Rayleigh};
2. the scattering coefficient of particles or aerosol, b_{scat};
3. the light absorption by gases, $b_{\text{abs-gas}}$; and,
4. the light absorption by the aerosol, b abs-aerosol.

Of these four, the scattering due to aerosol is usually assumed to dominate in haze.[2] The process of scattering amounts to the removal of light from the original beam and its redistribution in different directions. Scattering thus differs from absorption in which the light energy is lost to the absorber; in scattering, the light energy is only spatially redistributed. Nonetheless, this removal of light from the beam (or line of viewing) does result in extinction. It suffices to state only the final result in equation 3-2 for the usual assumption of 2% contrast threshold for an "average" human eye.[2,3]

$$L_v = \frac{3.9}{b_{\text{scat}}} \qquad (3\text{–}3)$$

Here, L_v is the visual range in meters and b is, as before, the extinction coefficient per meter along the path of sight for the case of a black object. If b is determined at a point without knowledge of the entire sight path, then L_v is "equivalent visual range," i.e., the distance one could see if the extinction coefficient were constant along the sight path. A discussion of the dependence of extinction coefficient on the amount of atmospheric aerosol follows in Section F.

"Visual quality" as perceived by even the well-trained observer is not so easily described. Among the complications is the fact that particles responsible for urban haze scatter more light in a direction close to that of the original beam (so-called forward-scatter) and less light in a backward direction. Thus, the same haze may appear to be much more dense when looking toward the sun and less dense when looking away from the sun.

Besides this directional factor, there is also a wavelength (or color) dependence. Ångstrom,[8] Junge,[9] and others have shown that the extinction coefficient of hazes in general is inversely proportional to a power of wavelength:

$$b = \frac{1}{\lambda\alpha} \qquad (3\text{–}4)$$

where α has measured values of around 1.0 to 1.5. This relation indicates that blue light (of shorter wavelengths) will be scattered to a greater degree than red light (of longer wavelengths). It is for this reason that the sun's disc, when observed through a haze that is dense enough to permit such viewing, appears red, orange or even brown though light absorption is not necessarily occurring.

D. COMPLICATIONS AND LIMITATIONS IN THE VISIBILITY PROBLEM

1. Smoke Plumes

Conner and Hodkinson,[10] in tests on the optical properties of well-controlled experimental smoke, found that visual effects are not inherent properties of the plumes but vary with the background of the plume and with illuminating and viewing conditions. Variation was much greater with white plumes than with black. Tests conducted with trained smoke inspectors showed that their evaluations of non-black smoke plumes were significantly influenced by these variations.

At least two real difficulties exist in making any generalizations about visual aspects of smoke plumes. First, it is not possible to determine the mass emitted per unit time from a smokestack solely on the basis of visually perceived light scatter or absorption. The mass per unit time emitted from the stack and not the appearance or optical properties of the plume is pertinent to the eventual air composition, even though appearance may be aesthetically objectionable.

Secondly, although many meteorological mixing equations have been proposed, they cannot describe individual eddies of smoke as the plume disintegrates. The equations were meant for describing averages and not an instantaneous property such as the extinction of light in some particular eddy of smoke as determined by eye, perhaps with the aid of a Ringelmann Chart.

Because of these problems, the topics of plumes and plume optics are discussed only briefly in this chapter, and the presentation is concerned primarily with the aerosol produced after the initial meteorological mixing of the plume has occurred. The reader is referred for further information on plume optics to the study by Conner and Hodkinson.[10]

The Ringelmann number may provide an objective measurement of public sentiment regarding the disagreeable appearance of smoke plumes, although aesthetic aspects are difficult to quantify. Robinson [2] shows the assumptions and size distribution information that are necessary for relating plume opacity to the aerosol content of smoke.

The difficulties inherent in visual evaluation of smoke plumes do not eliminate the possibility of using such observations as an aid in controlling air pollution. In principle, the Ringelmann Chart should be useful in estimating the obscuring of vision by plumes and in setting limits to control the visibility degradation downwind from a source of particulate matter. In practice, however, the problem is extremely complex and requires extensive study to develop better techniques for measuring the contribution of individual plumes to visibility problems.

2. Natural Aerosols and Hazes

The general problem of natural particulate matter—from whatever source—must be considered. The oceans produce salt particles, trees produce terpenes that may result in organic particles,[11] forest fires make smoke, and so on. Man has little hope of controlling the quality of air that enters the urban areas from uninhabited lands. Nonetheless, these low-humidity aerosols sometimes cause dramatic reduction in the visual range. In order to properly evaluate the importance of natural aerosols, the visibility of the air mass should be determined before it enters a populated area.

3. Fogs

When the relative humidity exceeds approximately 70 percent, many types of particles exhibit deliquescent behavior and grow into fog droplets. Natural particles such as sodium chloride from the sea as well as many products of human activity can thus act as condensation nuclei (Chapter 2). The property of deliquescence and the relative humidity at which rapid and large change in particle size occurs are both very dependent on the chemical composition and original size of the particles. As a result, unless the chemical composition as a function of particle size is known for the aerosol, very little can be said about the relationship between visibility in even "thin" fog and the amount of material present as pollutant.[12]

Because little deliquescence occurs below 70 percent relative humidity, the relationships to be presented here will be *limited* to the range of humidity from 0 percent to 70 percent. In cases of higher humidity, it is possible to decrease the relative humidity of the air by heating it for optical evaluation of the amount of particulate matter, as described by Charlson *et al.*[13] This humidity limitation has already been adopted in California.[14]

E. THE LOW-HUMIDITY, WELL-AGED HAZE

1. Size Distribution

Recent advances in both theory and technology have resulted in a simplification of the description of well-aged aerosols. Junge,[9] Friedlander,[15] Whitby,[16] and others [17] have shown that aerosols in the lowest region of the atmosphere (troposphere), whether over urban areas or not, tend to have similar size distributions. Figure 3–1 shows several typical size distributions to illustrate this feature.

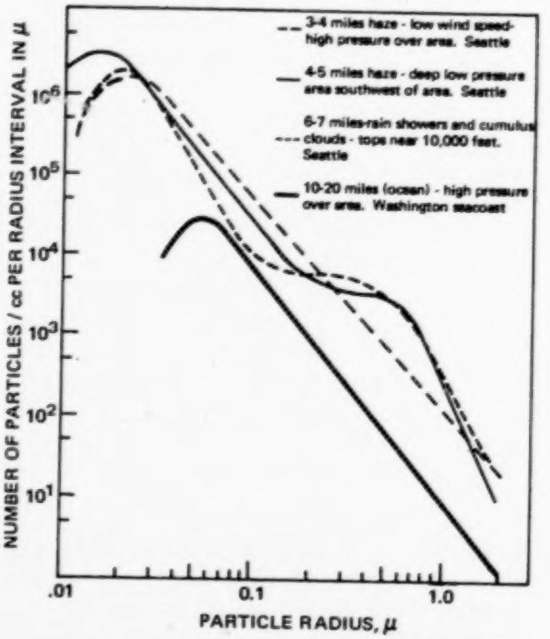

FIGURE 3–1. Four Typical Measured Size Distributions of Atmospheric Suspended Particles Together with the Corresponding Visibilities.[9] (The figure shows that aerosols in the lowest region of the atmosphere (the troposphere) tend always to have similar size distributions.)

If it is assumed that this recurring size distribution exists in general, then it is possible to relate the optical properties of the haze to the amount or mass concentration of material present. This generalization may not apply to freshly-formed smoke. Brief guidelines for determining when the size distribution has become sufficiently well-defined will be given later.

2. Mie Solutions

As mentioned earlier, another important assumption which is usually made is that, of the four extinction components, light scattering by aerosols dominates. Current research indicates that this is probably a justifiable assumption.[13] Figure 3–2 shows typical scattering cross sections for green light as a function of particle size for aerosol particles important in haze, computed via the theory of Gustav Mie.[18] For some particle sizes, differences in the scattering coefficient of a factor of three exist between the two refractive indices which span the realistic range for the atmospheric case. However, Pueschel and

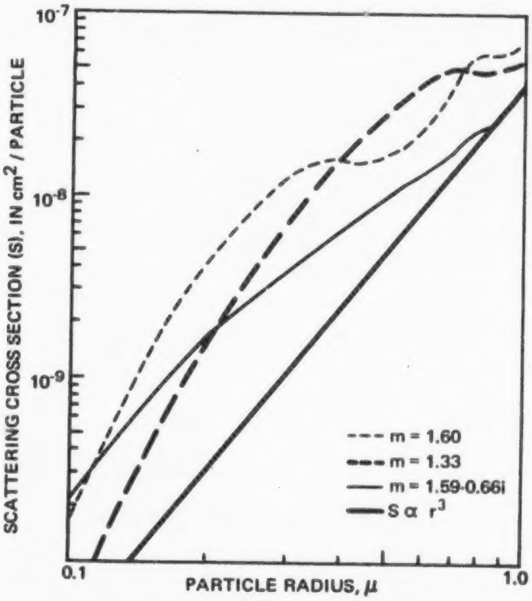

Figure 3–2. The Relation of Scattering Cross-Section to Particle Size for Particles of Three Different Refractive Indices.[18] (The figure shows that the scattering cross-section of atmospheric particles varies roughly as the cube of their radius within the range 0.1μ to 1.0μ. The range of refractive indices, 1.33 to 1.6, for which the proportionality holds includes most materials found in atmospheric aerosols.)

Noll [19] conclude that the extinction coefficient of aerosols in the troposphere is nearly independent of the refractive index of the particles if the size distribution is close to those described in Figure 3–1.

3. Dependence of Extinction on Particle Size for the Atmospheric Case

If the data in Figures 3–1 and 3–2 are broken down into narrow radius intervals (e.g., 0.01μ), and a calculation performed to yield the extinction coefficient for each radius interval, the particle size dependence of atmospheric extinction of green light is revealed. Figure 3–3 shows the results for a typical size distribution of spherical particles having a refractive index of 1.5.

In general, this procedure shows that a narrow range of particle sizes, usually from 0.1μ to 1μ radius controls the extinction coefficient and therefore the visibility. If the values of the extinction coefficient for each radius interval are then summed, the total extinction coefficient, due to scatter, b_{scat}, is obtained.

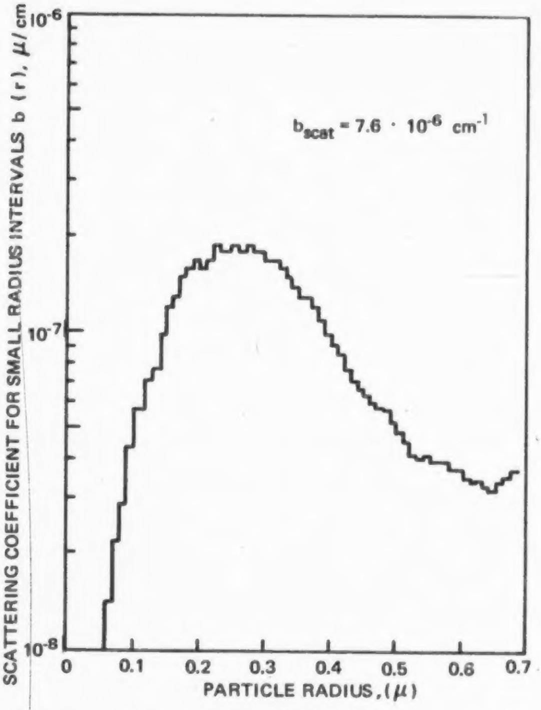

Figure 3–3. Cross Section Curve for a Typical Atmospheric Size Distribution.[19] (The figure shows the particle size dependence of atmospheric extinction obtained by breaking down the data of Figures 3–1 and 3–2 into 0.01μ-radius intervals, to yield the extinction coefficient of each radius interval.)

4. The Mass-Light Scattering Relationship

It is also possible to calculate the volume of particulate matter per unit volume of air (i.e., in cubic microns of aerosol per cubic centimeter of air). The familiar quantity of areosol mass concentration ($\mu g/m^3$) is proportional to this volume ratio via the particle density. Figure 3–4 shows the calculated relationship of aerosol volume (μ^3/cm^3) to scattering coefficient per meter (for green light [5500Å] and for a refractive index of 1.6) based on 32 individual measured size distributions. Sixteen of these size distributions used in Figure 3–4 were measured in Seattle under varying meteorological conditions. The remaining 16 were obtained in the Austrian Alps under conditions where, presumably,

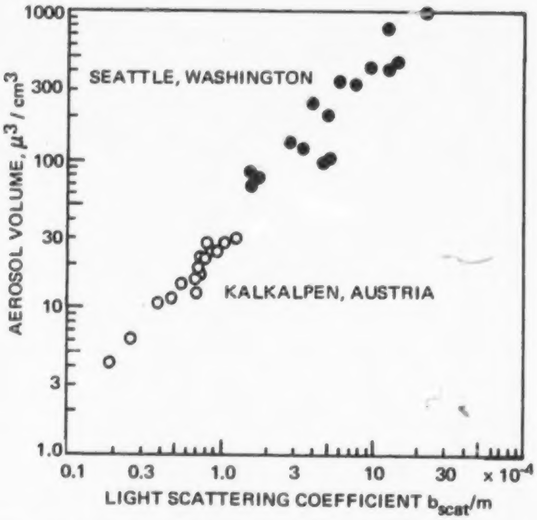

FIGURE 3–4. The Dependence of Scattering Coefficient (m^{-1}) on Volume of Aerosol Particles (μ^3/cm^3) Calculated from Measured Size Distributions.[19] (The solid circles are based on Seattle, Washington, data and the open circles on Kalkalpen, Austria, data.)

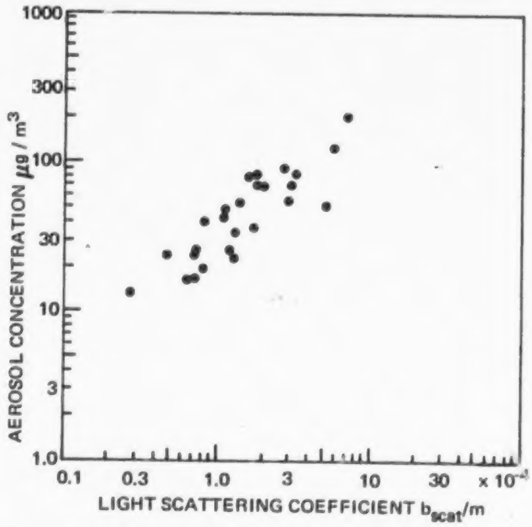

FIGURE 3–5. Measured Dependence of Mass of Aerosol Particles per Volume ($\mu g/m^3$) on the Light Scattering Coefficient (m^{-1}) in Seattle, November-December 1966.[19]

only natural aerosol was present. The implication of these calculations is that the volume, and thus the mass concentration of well-aged aerosol, is approximately proportional to the light scattering coefficient for atmospheric aerosols originating naturally or as the result of man's activity.[13]

It follows, therefore, that even though the aerosol mass is distributed over perhaps two or three decades of size, and light scatter is caused by particles of one narrow size range, a proportionality can exist between the number of particles scattering light and the total mass concentration of particles. Constant shape of the size distribution here implies that for all radius intervals, the number of particles in an interval is proportional to the number in the corresponding interval in a reference size distribution.

Figure 3–5 shows data obtained by an integrating nephelometer [13] for a wavelength of 5000 Å confirming this calculated dependence. The relationship can be summarized as follows:

$$\text{mass } (\mu g/m^3) \simeq 3\times 10^5 \text{ } b_{scat}/m. \qquad (3\text{–}5)$$

Figure 3–6 shows data like those of Figure 3–5 plotted in a different fashion to illustrate their distribution.[20] From equations 3–3 and 3–5, the product of equivalent visual range and mass concentration ($L_v\times$conc) can be obtained.

Since

$$L_v = \frac{3.9}{b_{scat}} \qquad (3\text{–}3)$$

multiplying both sides of the equation by concentration gives

$$L_v \times \text{conc} = \frac{3.9\times \text{conc}}{b_{scat}} \qquad (3\text{–}3a)$$

The units on both sides of this equation are mass per area (e.g., $\mu g/m^2$). If we use the particular proportionality in equation 3–5, then:

$$L_v \times \text{conc} \simeq 1.2\times 10^6 \ \mu g/m^2 = 1.2 \ g/m^2. \qquad (3\text{–}6)$$

This product has a simple physical significance: it is the mass of material in a column of length L_v and one square meter in cross section. In other words, it is the amount of material per square meter between the observer and a point at the limit of visibility. Each point of Figure 3–5 can be used to form this number since each pair of values of mass concentration and scattering coefficient can

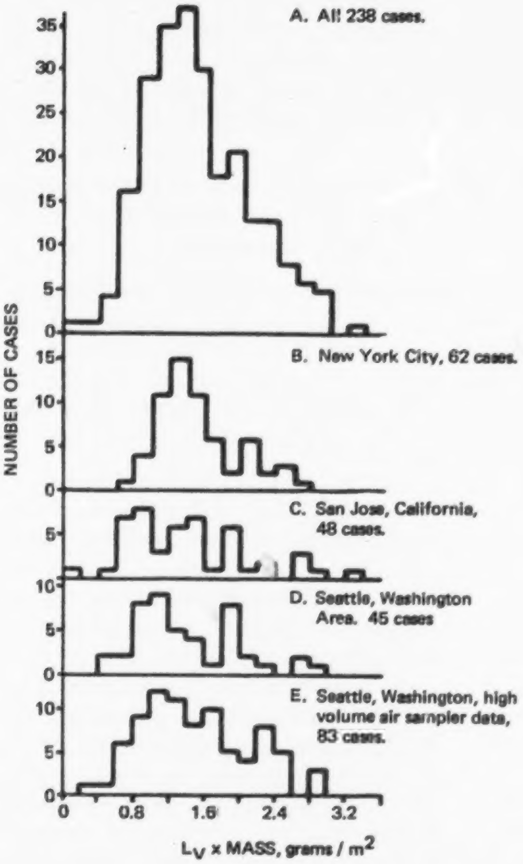

FIGURE 3–6. Histogram of Equivalent Visual Range-Mass Concentration Product at Several Locations.[20] (The figure shows the number of cases with a given range of values of the product of mass and equivalent visual range for data at various locations studied. This product represents the mass required to determine the visibility in a column one square meter in cross section along the light path.)

be used to obtain a different proportionality of the sort shown in equation 3–5. Figure 3–6 consists of histograms showing the number of occurrences for different values of the quantity $L_v \times$ conc found at various locations. Here, since the modal value is about 1.2 g/m², one can write:

$$L_v \times \text{conc} \simeq 1.2^{+1.2}_{-0.6} \ (\text{g/m}^2) \qquad (3\text{–}7)$$

to include virtually all cases.

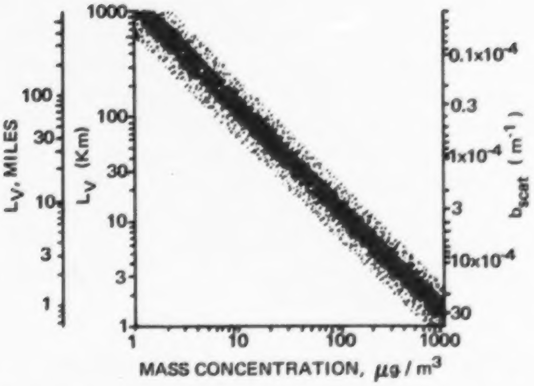

FIGURE 3–7. Relation Between Visual Range and Mass Concentration.[23] (This figure shows the inverse proportionality between visual range and mass concentration described by equations 3–7 and 3–8.)

Equation 3–5 can also be rewritten to include these upper and lower limits:

$$\text{mass } (\mu\text{g/m}^3) \simeq (3^{+3.0}_{-1.5}) \times 10^5 \ b_{scat}/\text{m}. \quad (3\text{–}8)$$

Data in essential agreement with this result have also been obtained recently by visual methods in Oakland, California.[21] As the data indicate that equation 3–8 is applicable in a wide variety of cases,[20] and because these locations seem to include a wide variety of air pollution character, it is anticipated that this generalization may be useful in many urban areas. The relationship is shown in Figure 3–7 and Table 3–1.[22] However, experimental verification of the mass-light extinction relationship is desirable for each location in question.

Table 3–1.—RELATION BETWEEN EQUIVALENT VISUAL RANGE AND PARTICLE CONCENTRATION.[19]

Mass concentration μg/m³	Scattering coefficient due to aerosol, b_{scat}/m	Equivalent visual range, km	Equivalent visual range, miles
10^{+10}_{-5}	0.3×10^{-4}	120.0	75.00
30^{+30}_{-15}	1.0×10^{-4}	40.0	25.00
100^{+100}_{-50}	3.3×10^{-4}	12.0	7.50
300^{+300}_{-150}	10.0×10^{-4}	4.0	2.50
1000^{+1000}_{-500}	33.0×10^{-4}	1.2	0.75

The accuracy of equations 3–7 and 3–8 depends on many factors and assumptions. This research area is an active one, and new data are constantly being acquired. Improvements in the understanding of these phenomena and the accuracy of descriptions will no doubt occur in the near future. However, these values are known to be accurate to well within one significant figure for the cases measured, and their utility for criterion purposes is therefore clear.

Photochemically produced aerosols, as well as other organic aerosols, can be expected to have similar visibility effects if their size distribution remains reasonably constant. Since the physical processes governing size distribution are assumed to be largely independent of the chemical nature of the particles,[15] the size distribution should be similar to those shown in Figure 3–1. However, if such particles are volatile or metastable to oxidation as suggested by Goetz,[23] the mass determination may be difficult. Although visibility degradation is evident in photochemical smog, experimentally determined size distribution data are needed.

It can be seen from Figure 3–5 that the practical unit of light scattering coefficient for 5000 Å wavelength is $10^{-4}/\text{m}$. The value $1 \times 10^{-4}/\text{m}$ represents fairly clean air with a particle mass concentration of about 30 $\mu\text{g/m}^3$, while [illegible]0 $\times 10^{-4}/\text{m}$ represents more polluted air (concentration about 300 $\mu\text{g/m}^3$). Of course, the reciprocal relationship (equation 3–3) could be used in combination with equation 3–5.

$$L_v\ (\text{Km}) \simeq 1.2_{0.6}^{2.4} \times 10^3/\text{conc}\ (\mu\text{g/m}^3). \qquad (3\text{–}9)$$

(See also Figure 3–7 and Table 3–1.) However, reciprocal relationships are harder to visualize than are direct proportionalities, and the extinction coefficient due to scatter (or just scattering coefficient) itself can serve as an index for particulate pollution.

It is interesting to compare the results of equation 3–7 with Robinson's[2] calculation of 0.34 g/m^2 for an oil aerosol in which all particles have the same 0.6μ diameter. Since this estimate represents a case in which all the particles are involved in the light scattering, it is not surprising that a mass smaller than that suggested by equation 3–7 is necessary for determining the visibility. In the atmosphere, a large percentage of the mass of the particulate matter is outside the size class important for light scatter (see Figures 3–1 and 3–3) and a somewhat higher mass per unit area is needed to determine the visibility.

F. DETERMINATION OF WELL-DEFINED CASES OF MASS-VISIBILITY RELATIONSHIPS

As discussed above, any generalization about the visibility-particle (aerosol) concentration relationship is dependent on a well-defined and nearly constant size distribution. The following list summarizes the cases in which equation 3–8 applies:

1. the relative humidity should be below 70 percent. (For a discussion of the relationship between visibility and concentrations of sulfur dioxide at various relative humidities, see a companion volume to this document. *Air Quality Criteria for Sulfur Oxides.*) Absolute humidity is relatively unimportant since the interaction of water vapor and hygroscopic aerosols depends mainly on relative humidity. In the case of a particularly hygroscopic aerosol, the 70 percent figure may be unreliable. Table 3–2 shows a list of compounds and the approximate humidity at which they deliquesce. If such a substance is present as a large percentage of the total aerosol, even though the rest of the following conditions may hold, the applicability of equation 3–5 might be open to doubt.
2. the size distribution must be well-established and close to the recurring form discussed earlier. Little experimental information is available on the length of time required for various types of fumes to attain a reasonably well-defined size distribution by coagulation and sedimentation. Measurement of this parameter is therefore desirable before any generalizations are made about visibility. The

Table 3-2.—THE RELATIVE HUMIDITY AT WHICH PHASE CHANGE OCCURS IN SOME DELIQUESCENT AEROSOLS.[24]

Substance	Approximate percent relative humidity at which phase change occurs at 25°C
Sodium hydroxide	7
Calcium chloride	29
Sulfuric acid	35
Magnesium chloride	33
Sodium iodide	38
Magnesium nitrate	53
Sodium bromide	58
Potassium iodide	69
Sodium nitrate	74
Sodium chloride	75
Potassium bromide	80
Potassium chloride	84
Barium chloride	90

most important qualification appears to be that the aerosol in question must not be freshly formed as in a smoke plume. Times of the order of one hour may be adequate for the establishment of a well-defined size distribution from a combustion-produced fume.

3. if visual observations are used, the line of sight cannot pass through smoke plumes or freshly formed clouds of fumes. As mentioned earlier, the light extinction coefficient is a spatial variable and thus the mass inferred by visual methods represents an average over a large distance. If the optical quantity (i.e. extinction coefficient due to scatter) is to be related to concentration measurements made at one point in space, then it should be determined at the same point. To illustrate the possible magnitude of the visibility variation, Figure 3-8 shows the variation of scattering coefficient measured with an integrating nephelometer across the city of Seattle on three days with different meteorological conditions. The results also emphasize the hazards inherent in visual observations.[25]

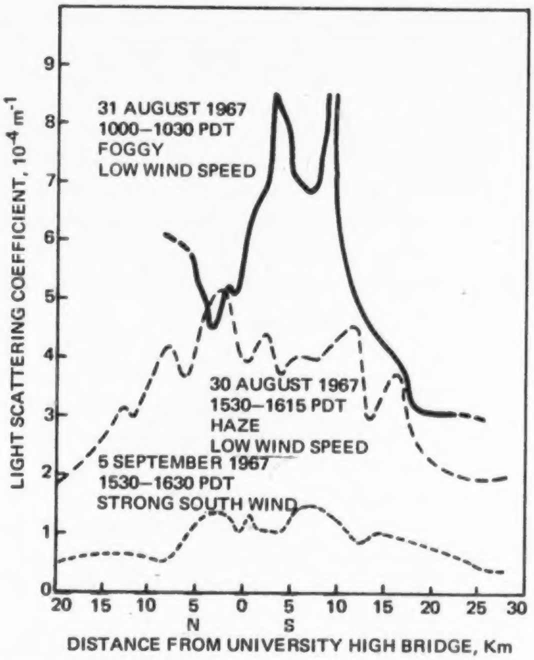

FIGURE 3-8. Three Horizontal Profiles Through the City of Seattle Taken Under Differing Meteorological Conditions.[25] (The figure illustrates the possible magnitude of variations in visibility.)

G. METHODS FOR DETERMINING LIGHT SCATTERING COEFFICIENT-MASS CONCENTRATION RELATIONSHIP

Methods for determining the extinction coefficient and/or the visibility (as related through equation 3-3) are not as well-established as for many meteorological quantities (e.g. wind, temperature) or for air pollution quantities (dustfall, mass concentrations, etc.). Three basic approaches can be differentiated:

1. the method of choice is an instantaneous point measurement of the extinction coefficient. The extinction coefficient due to scatter (which is assumed to dominate) can be measured with an integrating nephelometer such as was used by Charlson *et al.*[13, 25, 26]
2. the next most desirable methods involve the measurement of total extinction coefficient using light trans-

mission. Telephotometers of the shortest possible base line might be used, but once again have poor sensitivity for typical urban haze. Specially devised instruments have been designed and used with great difficulty in the range of extinction found in cities. Typical airport transmissometers are designed to be useful in fog and are thus of little value except in cases of extreme pollution and fog.

3. visual methods, though frequently used, should be avoided because of the subjectivity of the eye as a sensor and the variations between observers, as well as the spatial variation problem mentioned above. If it is necessary to use visual observations, the observers must be methodical and should use the rules adopted by the United States Weather Bureau.[27]

Ordinary methods (i.e. the high-volume air sampler) for the determination of mass concentration ($\mu g/m^3$) may suffice if care is taken to eliminate spurious large particles such as insects, etc. As recent data taken in Seattle show, newer sampling methods using newer types of filters are becoming available and allow a much shorter sampling time.

H. SUMMARY

The visual range, sometimes called the "visibility," is reduced by particulate matter in the air. It refers to the distance at which it is just possible to perceive and object against the horizon sky. Both the attenuation of light from the object and illumination of the air between the object and the observer tend to reduce visibility, since they reduce the perceived contrast between the object and its background. Attenuation of light passing through the air results from two optical effects which air molecules and small particles have on visible radiation: (1) the absorption of light energy and (2) the scattering of light out of the incident beam. In general, reduced visibility is primarily a result of scattering due to particulate matter. The "extinction coefficient," b_{scat}, is a measure of the degree of scattering, and it is related to the visual range of a black object as follows:

$$L_v = \frac{3.9}{b_{scat}} \text{ (m)} . \qquad (3\text{–}3)$$

Suspended particulates found in the atmosphere cover a broad range of size; however; the visibility is affected by a relatively narrow segment of this size distribution, usually from about 0.1μ to 1μ radius. Once particulate matter has been suspended in the air for some time, the distribution of particles by size tends to take on a typical pattern. Because of this, and because the visible light scattering is caused primarily by particles of one narrow size range, the scattering can be empirically related to the particulate concentration. This relationship is as follows:

$$L_v \simeq \frac{A \times 10^3}{G'} \qquad \text{(from 3–9)}$$

where G' = particle concentration ($\mu g/m^3$)
L_v = equivalent visual range
$A = 1.2^{2.4}_{0.6}$ for L_v expressed in kilometers and
$0.75^{1.5}_{.38}$ for L_v expressed in miles.

The ranges that are shown for the constant, A, cover virtually all cases studied. Deviations from equation 3–9 would be expected to occur when the relative humidity exceeds 70 percent, since many particles exhibit deliquescent behavior and grow into fog droplets. (For a discussion of the relationship between visibility and sulfur dioxide concentrations at various relative humidities, see a companion volume to this document, *Air Quality Criteria for Sulfur Oxides.*) Particles composed of sodium chloride, for example, would act as condensation nuclei and show rapid and large changes in size under such circumstances. Also, this relationship may not hold for photochemical smog, since it is not known whether its size distribution conforms to the necessary pattern.

Equation 3–9 provides a convenient means for estimating the expected visibility for different levels of particulate concentrations, under the conditions stated. With a typical rural concentration such as 30 $\mu g/m^3$, the visibility is about 25 miles; for common urban concentrations, such as 100 $\mu g/m^3$ and 200 $\mu g/m^3$, the visibility would be 7.5

miles and 3.75 miles, respectively. In addition to aesthetic degradation of the environment, reduced visibility has many consequences for the safe operation of aircraft and motor vehicles. When airports have heavy traffic, visibility below 5 miles tends to slow operations, since it is necessary to maintain larger distances between aircraft. Federal air regulations prescribe limitations on aircraft operating under conditions of reduced visibility; they become increasingly severe as the visibility decreases from 5 miles (150 $\mu g/m^3$) to 3 miles (250 $\mu g/m^3$) to one mile (750 $\mu g/m^3$). Based on the empirical variations in equation 3–9, the same visibilities could occur under certain circumstances, at concentrations one-half of these calculated values. Thus, a concentration of 75 $\mu g/m^3$ might produce a visibility of 5 miles in some instances.

I. REFERENCES

1. Holzworth, G. C. "Some Effects of Air Pollution on Visibility in and near Cities." In: Air Over Cities Symposium, U.S. Dept. of Health, Education, and Welfare, Robert A. Taft Sanitary Engineering Center, Cincinnati, Ohio, Technical Report A62–5, 1961, pp. 69–88.
2. Robinson, E. "Effects of Air Pollution on Visibility." In: Air Pollution, Chapt. 11, Vol. 1, 2nd edition, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 349–400.
3. Middleton, W. E. K. "Vision Through the Atmosphere." University of Toronto Press, 1952.
4. "Federal Aviation Regulations." Federal Aviation Agency, 1967.
5. "A Study of Pollution—Air." A staff report to the Committee on Public Works, U.S. Senate, Washington, D.C., 1967.
6. "New York-New Jersey Air Pollution Abatement Activity, Phase II—Particulate Matter." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Washington, D.C., 1967.
7. "Kansas City, Kansas-Kansas City, Missouri, Air Pollution Abatement Activity, Phase II—Pre-conference Investigations." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, Washington, D.C., March 1968.
8. Ångstrom, A. K. "On the Atmospheric Transmission of Sun Radiation II." Geograph. Ann. (Stockholm), Vol. 12, pp. 139–159, 1930.
9. Junge, C. E. "Air Chemistry and Radioactivity." Academic Press, New York, 1963.
10. Conner, W. D. and Hodkinson, J. R. "Optical Properties and Visual Effects of Smoke-Stack Plumes." U.S. Dept. of Health, Education, and Welfare, National Center for Air Pollution Control, PHS–Pub–999–AP–30, 1967.
11. Went, F. W. "Dispersion and Disposal of Organic Materials in the Atmosphere." Preprint Series 31, University of Nevada, Desert Research Institute, 1966.
12. Pilat, M. J. and Charlson, R. J. "Theoretical and Optical Studies of Humidity Effects on the Size Distribution of a Hygroscopic Aerosol." J. Rech. Atmospheriques, Vol. 2, pp. 165–170, 1966.
13. Charlson, R. J., Horvath, H., and Pueschel, R. F. "The Direct Measurement of Atmospheric Light Scattering Coefficient for Studies of Visibility and Air Pollution." Atmos. Environ., Vol. 1, pp. 469–478, 1967.
14. "California Standards for Ambient Air Quality and Motor Vehicle Exhaust—Technical Report." Dept. of Public Health, Berkeley, California, 1960.
15. Friedlander, S. K. and Wang, C. S. "The Self-Preserving Particle Size Distribution for Coagulation by Brownian Motion." J. Colloid Interface Sci., Vol. 22, pp. 126–132, 1966.
16. Whitby, K. T. and Clark, W. E. "Electric Aerosol Particle Counting and Size Distribution Measuring System for the 0.15μ to 1μ Size Range." Tellus, Vol. 18, pp. 573–586, 1966.
17. Peterson, C. M. and Paulus, H. J. "Micrometeorological Variables Applied to the Analysis of Variation in Aerosol Concentration and Size." Preprint. (Presented at the 60th Annual Meeting, Air Pollution Control Association, Cleveland, Ohio, June 11–16, 1967.)
18. Mie, G. "Beiträge zur Optik trüber Medien, Speziell Kolloidaler Metallosungen." Ann. Physik, Vol. 25, pp. 377–455, 1968.
19. Pueschel, R. F. and Noll, K. E. "Visibility and Aerosol Size Frequency Distribution." J. Appl. Meteorol., Vol. 6, pp. 1045–1052, 1967.
20. Charlson, R. J., Ahlquist, N. C., and Horvath, H. "On the Generality or Correlation of Atmospheric Aerosal Mass Concentration and Light Scatter." Atmos. Environ., Vol. 2, pp. 455–464, 1968.
21. Noll, K. E., Mueller, P. K., and Imada, M. "Visibility and Aerosol Concentration in Urban Air." Atmos. Environ., Vol. 2, pp. 465–475, 1968.
22. Charlson, R. J. "Atmospheric Aerosol Research at the University of Washington." J. Air Pollution Control Assoc., Vol. 18, pp. 652–654, 1968.
23. Goetz, A., Preining, O., and Kallai, T. "The Metastability of Natural and Urban Aerosols." Rev. Geofis. Pura Appl. (Milano), Vol. 60, pp. 67–80, 1961.
24. Acheson, D. T. "Vapor Pressures of Saturated Aqueous Salt Solutions." Proc. Intern. Symp. Humidity and Moisture, Washington, D.C., May 1963, pp. 521–530.

25. Ahlquist, N. C. and Charlson, R. J. "Measurement of the Vertical and Horizontal Profile of Aerosol Concentration in Urban Air with the Integrating Nephelometer." Environ. Sci. Technol., Vol. 2, pp. 363–366, 1968.

26. Ahlquist, N. C. and Charlson, R. J. "A New Instrument for Evaluating the Visual Quality of Air." J. Air Pollution Control Assoc., Vol. 17, pp. 467–469, 1967.

27. "Manual of Surface Observations (WBAN)." Circular N, 7th Edition (Revised to include changes 1 through 14), U.S. Weather Bureau, January 1968.

Chapter 4

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON MATERIALS

Table of Contents

Page
A. INTRODUCTION 65
B. EFFECTS OF PARTICULATE MATTER ON METALS 65
C. EFFECTS OF PARTICULATE MATTER ON BUILDING MATERIALS 69
D. SOILING AND DETERIORATION OF PAINTED SURFACES 71
E. SOILING AND DEGRADATION OF TEXTILES 71
F. SUMMARY 72
G. REFERENCES 74

List of Figures

Figure
4–1 Plot of Rate of Rusting Versus Dustfall at Four Locations 66
4–2 Weight Loss from Zinc Specimen as a Function of Exposure Time 67
4–3 District Building, Washington, D. C. 67

List of Tables

Table
4–1 Corrosion of Open-Hearth Iron Specimens in Different Locations 66
4–2 Corrosivity of Atmospheres towards Steel and Zinc at Selected Locations Relative to that at State College 68
4–3 The Corrosion Rates of Metals in Various Locations 70

Chapter 4

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON MATERIALS

A. INTRODUCTION

Airborne particles may damage surfaces merely by settling on them. A light deposit of dust makes surfaces appear dingy, and the frequent cleaning necessary in a dusty atmosphere weakens materials and costs money.

Particles can also cause direct or chemical deterioration of materials. The nature and extent of the deterioration depends on the chemical activity of the particles in their environment and the relative susceptibility of the receiving material. Particles may cause chemical deterioration either by acting as condensation nuclei for the retention of adsorbed gases or harmful acids, or by their own innate corrosive action.

In the following sections it will be seen that particulate air pollution plays an important role in the corrosion of metals; in the soiling, damage, and erosion of coatings and painted surfaces, building stones, marble, and other building materials; and in the soiling and degradation of textiles.

B. EFFECTS OF PARTICULATE MATTER ON METALS

Atmospheric particles may accelerate the corrosion of iron, steel, and various nonferrous metals.

Metals are generally resistant to attack in dry air,[1] and even clean moist air does not cause significant corrosion.[1, 2] Furthermore, inert dust and soot particles without sulfur compounds as constituents do not of themselves cause marked corrosion.[1, 3] Particles may, however, contribute to accelerated corrosion in two ways.[4] First, they may be intrinsically active, and secondly, although inactive, they may be capable of absorbing or adsorbing active gases (such as SO_2) from the atmosphere.

Those particles which are inactive and have negligible capacity for absorption or adsorption have little effect other than that of acting as droplet nuclei in the atmosphere. For example, silica particles do not accelerate the rate of metal corrosion even in the presence of SO_2. On the other hand, charcoal (carbonaceous) particles in atmospheres with relative humidities below 100 percent cause a large increase in the rate of corrosion in the presence of SO_2 traces, presumably through the local concentration of the gas by adsorption.[4] The laboratory research which led to these findings was based on particulate concentrations of 0.4 mg/cm^2 (equivalent to 0.3 tons/mi^2) and abnormally high SO_2 concentrations of 100 ppm.

Active hygroscopic particles such as sulfate and chloride salts and sulfuric acid aerosol serve as corrosion nuclei. Their presence in the atmosphere can initiate corrosion, even at low relative humidities.[5] Laboratory studies of bare and varnished steel test panels, properly polished and degreased, and then inoculated with finely divided particles of various substances such as are commonly found in the atmosphere, were conducted by Preston and Sanyal.[5] Particles of chloride, sulfate, chromate, and oxide salts, and boiler and flue dusts were used. The metal test panels were exposed to atmospheres of pure clean air and of air containing SO_2 at various humidities, and the resulting corrosion was measured. Filiform corrosion, characterized by a filamental configuration, the primary phase in electrolytic corrosion, was noted in all cases. Corrosion rates are low when the relative humidity is below 70 percent, but they increase at higher humidities.[4, 6] In most of the cases in this study, corrosion increased with increased humidity even in clean air. The addition of traces of

SO_2 to the test atmosphere greatly increased the rate of corrosion in all instances.[5]

Field experiments show that the rate of corrosion of various metals is accelerated in urban and industrial areas because of the greater atmospheric concentrations of both particulate matter and sulfur compounds. Standardized open-hearth iron specimens, after exposure for one year at a number of diverse locations throughout the world, were observed by Hudson[7] to have a manifiold variation in weight loss. Iron specimens exposed for one year to desert and arctic environments, the least polluted areas, were the least corroded, i.e., had the smallest losses in weight. Those speimens exposed at heavily-polluted urban industrial sites were the most corroded, more so than similar specimens exposed at many marine and tropical locations, Table 4–1. Hudson, Figure 4–1, also correlated the rate of rusting of mild steel specimens and dustfall levels for four diverse areas, from a heavily industrialized area in Sheffield, England, to a rural area (Llanwrtyd Wells). The iron specimens corroded four times as fast in the polluted industrial atmosphere as they did in the rural atmosphere.

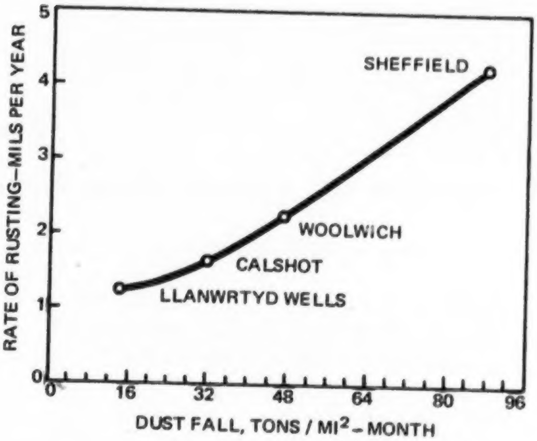

FIGURE 4–1. Plot of Rate of Rusting Versus Dustfall at Four Locations.[7] (The figure plots the rate of rusting of mild steel versus dustfall, and shows that corrosion is four times as rapid in an industrial area (Sheffield) as it is in the rural area (Llanwrtyd Wells)).

Committee B–3 of the American Society of Testing Materials (ASTM)[8, 9] studied corrosion rates of steel and zinc panels exposed for one year at several locations in the United States. The relative corrosivities of various atmospheres at 19 sites were compared to that of the rural site of State College, Pennsylvania, which was used as a base. This study (Table 4–2) confirms Hudson's observations that industrial locations with

Table 4–1.—CORROSION OF OPEN HEARTH IRON SPECIMENS IN DIFFERENT LOCATIONS.[7]

Location	Type of atmosphere	Annual weight loss, g	Relative corrosivity
Khartoum, Sudan	Dry inland	0.16	1
Abisko, North Sweden	Unpolluted	0.46	3
Aro, Nigeria	Tropical inland	1.19	8
Singapore, Malaya	Tropical marine	1.36	9
Basrah, Iran	Dry inland	1.39	9
Apapa, Nigeria	Tropical marine	2.29	15
State College, Pa., USA	Rural	3.75	25
Berlin, Germany	Semi-industrial	4.71	32
Llanwrtyd Wells, British Isles	Semi-marine	5.23	35
Calshot, British Isles	Marine	6.10	41
Sandy Hook, N.J., USA	Marine-semi-industrial	7.34	50
Congella, South Africa	Marine	7.34	50
Motherwell, British Isles	Industrial	8.17	55
Woolwich, British Isles	Industrial	8.91	60
Pittsburgh, Pa., USA	Industrial	9.65	65
Sheffield Univ., British Isles	Industrial	11.53	78
Derby South End, British Isles	Industrial	12.05	81
Derby North End, British Isles	Industrial	12.52	84
Frodingham, British Isles	Industrial	14.81	100

high concentrations of particles and of oxides of sulfur are more corrosive to steel and zinc than less industrialized areas. As shown in Table 4–2, steel specimens corroded in one year approximately 3.1 times as much in New York City (spring exposure) and 3.3 times as much in Kearny, New Jersey, as similar specimens at State College, Pennsylvania. Zinc specimens corroded 3.6 and 2.6 times as much in New York City (spring exposure) and Kearny respectively, as similar specimens exposed at State College. Both steel and zinc specimens in New York City (fall exposure when the particle and SO_2 concentrations in the atmosphere are greater than in spring) corroded 6.0 and 3.7 times as much respectively as similar steel and zinc speciments at Sate College.[9] The authors of the resulting papers did not give mean concentrations of suspended particulate matter for the various locations; but approximate concentrations, based on NASN measurements [10] made after the corrosion studies, are given here. Based on Table 4–3, a town the size of State College, Pa., could have a mean suspended particulate-matter level of 60 μg/m³ to 65 μg/m³. The mean suspended particulate matter concentrations were 176

Table 4–2.—CORROSIVITY OF ATMOSPHERES TOWARDS STEEL AND ZINC AT SELECTED LOCATIONS RELATIVE TO THAT AT STATE COLLEGE, PENNSYLVANIA.[9]

Location	Type of atmosphere	Relative corrosivity of one-year test for	
		Steel	Zinc
Norman Wells, N.W.T., Canada	Polar-Rural	0.03	0.4
Esquimalt, Vancouver Is., Canada	Rural-Marine [a]	0.5	0.4
Saskatoon, Sask., Canada	Rural	0.6	0.5
Perrine, Fla	Rural	0.9	1.0
State College, Pa	Rural	1.0	1.0
Ottawa, Canada	Semi-Rural	1.0	1.2
Middletown, Ohio	Semi-Industrial	1.2	.9
Trail, B.C., Canada	Semi-Rural	1.4	1.6
Montreal, Que., Canada	Industrial	1.5	2.2
Halifax, N.S., Canada	Rural-Marine	1.5	1.6
South Bend, Pa	Semi-Rural	1.5	1.5
Kure Beach, N.C., 800-ft site	Marine (800 ft from ocean)	1.8	1.7
Point Reyes, Calif	Marine	1.8	1.8
Sandy Hook, N.J	Industrial-Marine	2.2	1.6
New York, N.Y. (spring exposure)	Industrial	3.1	3.6
Kearny, N.J	Industrial	3.3	2.6
Halifax, N.S., Canada	Industrial-Marine [b]	3.8	18.0
New York, N.Y. (fall exposure)	Industrial	6.0	3.7
Daytona Beach, Fla	Marine	7.1	2.6
Kure Beach, N.C., 80-ft site	Marine (80 ft from ocean)	13.0	5.7

[a] While test site is 1500 ft. from brackish water, prevailing winds are from inland and prevent deposition of salt water spray.

[b] Test site is near a smokestack; prevailing winds blow fumes over the test site.

Table 4–3.—THE CORROSION RATES OF METALS IN VARIOUS LOCATIONS.[9]

Test Site	Corrosion rate, mil/year		
	Nickel 200	Monel alloy 400	Copper
Altoona, Pa. (Heavy industrial-R.R.)	0.222	0.076	0.055
New York, N.Y. (Urban-industrial)	0.144	0.062	0.054
State College, Pa. (Rural-farm)	0.0085	0.007	0.017
Phoenix, Ariz. (Rural-semiarid)	0.0015	0.002	0.005

$\mu g/m^3$ in New York City and 131 $\mu g/m^3$ at Elizabeth, New Jersey, near Kearney,[10] it should be pointed out that there are significant differences in levels of gaseous pollution, particularly sulfur dioxide, between State College and the larger industrial communities. Consequently, the differences in corrosion rates cannot be solely attributed to the effects of particulates.

In Chicago and St. Louis, steel panels were exposed at a number of sites, and measurements were taken of corrosion rates and of levels of sulfur dioxide and particulates.[11] In St. Louis, except for one exceptionally polluted site, corrosion losses correlated well with sulfur dioxide levels, averaging 30 percent to 80 percent higher than corrosion losses measured in nonurban locations. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO_2 level of 0.03 ppm). Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Although suspended particulate levels measured in Chicago with high-volume samplers correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations had the dominant influence on corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 percent to 17 percent weight loss in steel panels) at annual average sulfur dioxide concentrations in the range of 0.03 ppm to 0.12 ppm, and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Comparative studies of the rates of corrosion of zinc-coated steel panels exposed to various community atmospheres were conducted by Committee B-3 of the ASTM.[9] The corrosion rates of zinc at four of the locations over a six-year period are shown in Figure 4-2, as weight loss versus time. For each location, the corrosion rate is essentially constant with time. The atmosphere of New York City with greater concentration of particles and sulfur oxides produces a steeper rate of corrosion of zinc than that of Kearny, New Jersey, which is also heavily polluted. Zinc corrodes at a greater rate in both industrial communities than in the rural or semirural sites in South Bend, Pennsylvania, and State College, Pennsylvania.[9]

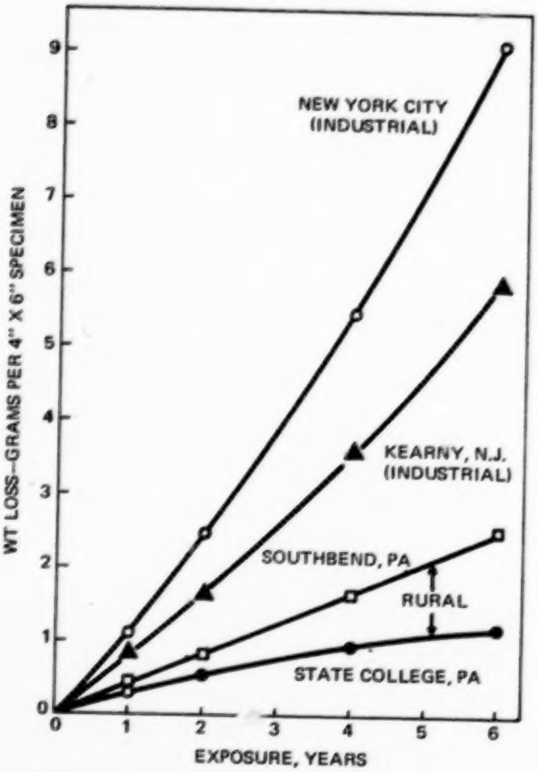

FIGURE 4–2. Weight Loss from Zinc Specimen as a Function of Exposure Time.[9] (The figure shows the rate of corrosion of zinc at four locations in the United States, and indicates that corrosion is more rapid in industrial areas.)

Studies of the effects of air pollution on the atmospheric corrosion of three metals (nickel 200, Monel alloy 400, and copper) exposed to four diverse atmospheres (heavy-industrial, urban-industrial, rural-farm, and rural-semiarid) were conducted over a 20-year period.[9] Nickel 200 is 99.5 percent nickel and Monel 400 is essentially 30 percent copper and 70 percent nickel. Though these metals are relatively corrosion-resistant, it will be noted from Table 4-3 that they are corroded more in industrial atmospheres than in rural ones. However, because of the corrosion resistance of the metals, all of the rates are relatively low when compared to those for unalloyed iron or steel. In the industrial locations, nickel 200 corroded at rates two to four times greater than Monel (nickel-copper) alloy 400, while in the rural locations, its corrosion rate was about equal to that of Monel alloy 400, and only one-half the rate for copper. Even Monel 400, with its superior corrosion resistance, when used as a gutter in an industrial installation, became pitted when the soot was permitted to accumulate.[9] Unburned carbon in the soot led to the formation of local galvanic corrosion cells. The result was premature perforation and accelerated attack of the metal sheet.

Larrabee[12] confirmed that the corrosion resistance of steel is greatly improved by the addition of small amounts of copper, or low alloys of chromium, nickel, copper, and phosphorus. The additional cost of such alloy steels when they are used to resist atmospheric corrosion is attributable to air pollution.

Particles can cause corrosion of electronic gear of all types even where pervious metals are used to minimize such corrosion. Electrical instruments and electronic components are factors of growing importance in the computer and missile industries, and also monitor and control an increasingly large share of manufacturing processes.[13] Oily or tarry particles, commonly found in industrial and urban areas, are serious factors in the corrosion and failure of electric contacts, connectors, and components.[13]

Dust can act mechanically or chemically on electric contacts. It can deposit on the surfaces and interfere with electrical contact closure, it can become imbedded in the surfaces of contacts, or it can induce wear by abrasion if the contacts slide.[13,14] Hygroscopic dusts, accumulated on contacts, will absorb water to form thin electrolytic films which are corrosive to base metals.[5] If the contact members are not of identical composition, galvanic corrosion may occur. The tarnish and corrosion films impair or cause failures in electrical conductivity, and can be avoided or minimized only by fabricating the electrical contacts out of nonreactive metals, by encapsulation, or by air purification devices such as filters or by gas-absorbing chemicals. Any of these solutions to the problem increases costs.

C. EFFECTS OF PARTICULATE MATTER ON BUILDING MATERIALS

Building materials and surfaces are soiled, disfigured, and damaged by atmospheric particles. Some of these stick to surfaces of stone, brick, paint, glass, and composition materials, forming a film of tarry soot and grit which may or may not be removed by the action of the rain. The result is a dingy, soiled appearance (Figure 4–3), a loss in aesthetic attractiveness and, in many cases, a physical-chemical degradation or erosion of these surfaces.

In cities where large quantities of soot-providing fuel are burned, the problem is particularly severe. Much money and effort have been spent on sandblasting off the sooty layers which have accumulated on prominent buildings in burning soot-producing fuel cities.[15–17] The tarry substances or carbonaceous material resulting from inefficient combustion of soot-producing fuel are likely to be sticky and also acidic;[2] if not flushed off by rain they will adhere to surfaces and corrode them over extended periods of time.[18] Smoke particles may also act as a reservoir for adsorbed gases, such as SO_2, and harmful acids, including sulfurous, sulfuric, and hydrochloric acids, as well as hydrogen sulfide. These materials cause the deterioration of many of the less resistant tyes of masonry.[19,20]

Buildings in polluted areas become unsightly quickly and require periodic cleaning and maintenance to remove the tarry asphalt-like deposits. In Washington D.C. it was found necessary to clean the smoke and grime from the new Supreme Court building even before initial occupancy (in the mid-thirties).[16] While the soiling effect of soot is by far the most evident to the eye, it does not in itself cause the deterioration of building material. The destruction is due to acids alone or to acids adsorbed on particulate matter.

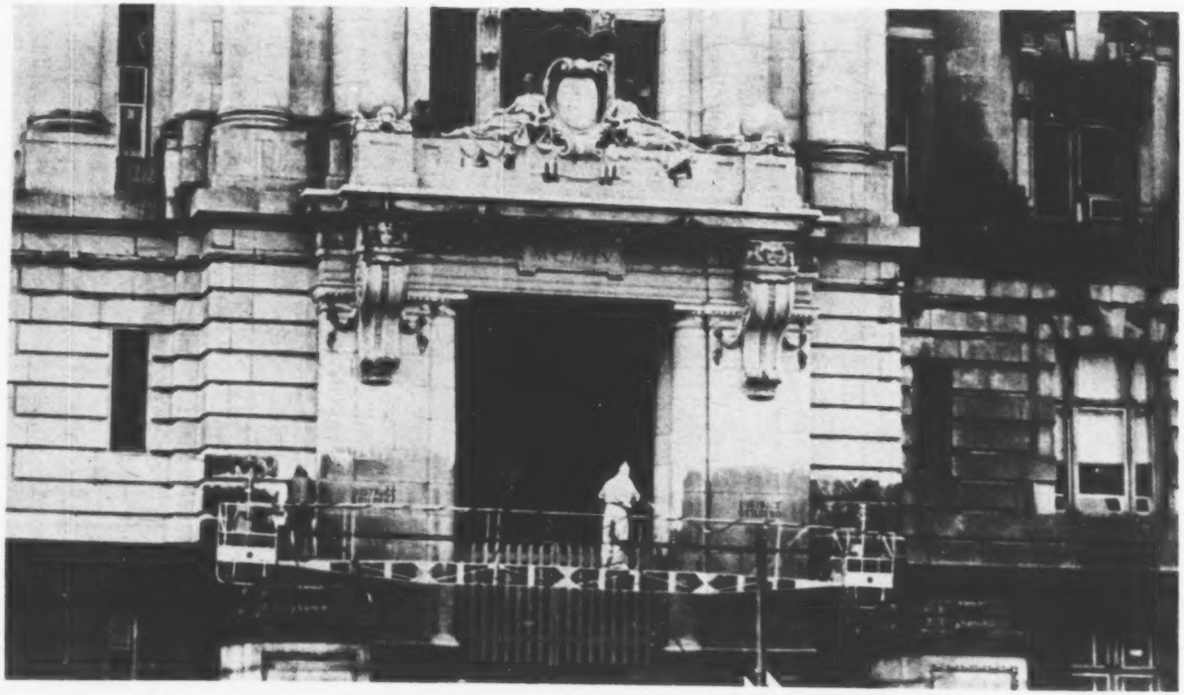

FIGURE 4–3. District Building, Washington, D.C. (This photograph shows sooty layers deposited on a public building, and the effect of sandblasting on restoring appearance. The photograph was taken on October 26, 1960.)

D. SOILING AND DETERIORATION OF PAINTED SURFACES

Painted surfaces, walls, and ceilings of homes and other buildings are soiled by tarry and other particulate substances in the atmosphere. Furthermore, there are both liquid and solid particles present in polluted air in the form of fumes and mists of varying chemical composition which react with painted surfaces.[18, 21] The damage done is both aesthetic and material, and may affect not only buildings but also paintings and other works of art.

Auto finishes have been observed to become pitted and stained by iron particles deposited on them from a metal grinding operation nearby,[6, 22] while chromic acid mist from an electroplating operation formed a brown stain on light colored cars and a "blushing" on darker shades of paint. Repainting was required because the color changes were not reversible by washing or polishing.[18, 23] Cars parked near demolition operations of brick buildings have been severely damaged by alkali mortar dust in the presence of moisture.[18] Characteristic pitting of painted surfaces is also caused by sodium carbonate particles from such industrial processing as soda ash manufacture.

Water soluble chlorides and sulfates—mostly the iron, copper, calcium, and zinc salts—are commonly found in particulate samples from cities [21, 23, 24] and in rainwater.[23] These water-soluble particles are potential sources of osmotic blistering of painted surfaces. The effects on weathering by small quantities of such particles have been examined [21] during laboratory studies of accelerated aging of various paint panels. The presence of 0.1 ppm of iron in the water in the accelerated weather apparatus produced yellow staining, while 0.5 ppm of copper produced severe brown staining. These same effects may be expected on natural exposure.[21]

Dust particles settling on wet varnish and paint films produce visible imperfections and reduction in the electrical resistance and anticorrosive properties of such films.[25] In laboratory tests to examine the relationship to corrosion of dust particles under the protective coatings, two sets of metal panels were coated with a varnish formulation under a "dust-free" apparatus, and then one set was removed from the apparatus so that the varnish films dried in contact with the laboratory air. The panels were then immersed in 3 percent aqueous sodium chloride for seven and one-half months. Where the varnish film had dried in "normal" laboratory room air, considerable rusting occurred on the panels after six months of immersion; no corrosion occurred on the panels dried in the "dust-free" environment. Clearly the dust particles that settled on the films of the panels during the drying period were a predominant factor in initiating corrosion of the metal.[25]

The exteriors of buildings in industrial areas, when repainted, collect numerous black specks on their surfaces even before the paint has dried.[26] Within two to four years depending on the degree of particle concentration, these building exteriors are distinctly soiled and require repainting.[16, 26]

E. SOILING AND DEGRADATION OF TEXTILES

Soiling of clothing, curtains, and other textiles not only diminishes their aesthetic appeal but also reduces their functional effectiveness. A garment which soils readily will not have the same user appeal as one which does not, even though its performance may be equal in other respects. Economic costs are therefore involved both in the extra cleaning of garments which soil readily and in the development and manufacture of soil-resistant textiles.

The extent of soiling of textiles, such as curtains, is influenced by various external factors such as temperature, relative humidity, and wind speed and the specific size and characteristics of the atmospheric particles. In addition, the degree of soiling is dependent on the construction and finish of the textile material and the type of fiber from which it is made.[27]

When large particles are deposited on the surface of the textile, the soiling may be superficial. In moving air, however, the particles may be directly intercepted by the individual fibers or may be thrown onto the fibers as air sweeps through the intricate channels between them, i.e., the material behaves as a filter. The soiling of curtains and flags is an illustration of this effect. Airborne particles can also affect the cleanliness of yarns and fabrics during their manufacture. Economic loss to textile manufacturers can occur unless suitable air filters are used in manufacturing plants.[27] Laboratory studies by Rees,[27] using a dust circulating apparatus under controlled conditions; show that a closely woven fabric of low porosity best resists soiling by airborne particles.

If an exposed textile material acquires an electrostatic charge, for example by friction, and is able to retain its charge, charged particles of opposite sign will be attracted to it, thereby increasing its rate of soiling.[27] Cellulose acetate and some of the synthetic textiles acquire, by friction during spinning and weaving, electrostatic charges which, because of the high insulating properties of these materials, are retained for a long period of time. This results in troublesome "fog-marking," caused by attraction of airborne particles to the charged textile.[27]

In laboratory investigations of the electrostatic attraction of airborne particles to cotton textiles, Rees [27] found that soiling is more rapid when the fabric is charged than when uncharged, and that the rate of soiling is increased by raising the applied electric potential (which increases the charge density on the fabric). For any given potential, the rate of soiling appears to be greater for a positively charged fabric specimen than for a negatively charged one.

The vulnerability of textile fabrics and furnishings to the acid components of particulate matter depends on the chemical composition of the textiles.[28] Cellulosic fibers, such as cotton, linen, hemp, jute, and manmade rayon are particularly sensitive to attack from such substances as sulfurous and sulfuric acids, while animal fibers, such as wool and furs, are more resistant to acid damage.[28]

Curtains are particularly vulnerable to air pollutants and often deteriorate quickly hanging at open windows; soiling occurs to some extent even when windows are closed. The curtain material acts more or less as a filter for acid-laden dust and soot. Airborne metallic iron and zinc particles, constituents of city dust,[23, 28, 29] are catalysts and promote oxidation of sulfur dioxide to sulfuric acid, which may contribute to textile degradation. Curtains weakened by conditions of exposure arising partly from atmospheric soiling and acidity give way in a characteristic manner by splitting in parallel lines along the folds where the greatest number of particles accumulated.

F. SUMMARY

Airborne particles—including soot, dust, fumes, and mist—can, according to their chemical composition and physical state, cause a wide range of damage to materials. They may cause deterioration merely by settling on surfaces and soiling them thus creating a need for more frequent cleaning which in itself weakens materials. More importantly, they can cause direct chemical damage to materials in two ways: First, through their own intrinsic corrosiveness, and secondly, through the action of corrosive chemicals which they may have absorbed or adsorbed. Airborne particulates have been implicated in the corrosion of metals and metallic surfaces; in the soiling, damage, and erosion of buildings and other structures; in the discoloration and destruction of painted surfaces; and in the aesthetic degradation and damage of fabrics and clothing.

Metals ordinarily can resist corrosion in dry air alone, or even in moist clean air. Even inert dust or soot, in the absence of active chemical agents has little effect on metal surfaces. However, hydroscopic particles commonly found in the atmosphere can corrode metal surfaces although no other pollutants may be present. This was shown in laboratory studies in which steel test panels were dusted with various common particulates. Although corrosion rates were low at relative humidities under 70 percent, they tended to increase with increased humidity. The addition of sulfur dioxide to the laboratory air greatly accelerated the rates of corrosion.

In general, there is an increasing rate of corrosivity as we go from dry, unpopulated environments, the least polluted areas, to heavily polluted urban industrial sites. Samples of iron, for example, have shown weight loss due to corrosion that is four times greater in industrial atmospheres than in rural atmospheres. Steel samples corroded in one year 3.1 times as much in New York City (spring exposure), where the particulate concentration was 176 $\mu g/m^3$, as in the rural atmosphere of State College, Pennsylvania, where the mean concentration could be expected to range about 60 $\mu g/m^3$ to 65 $\mu g/m^3$. Steel samples exposed in New York in the fall of the year, when the particulate and sulfur dioxide levels are higher than in the spring, corroded 6 times faster than the samples at State College. Similarly, zinc samples exposed in New York corroded about 3.6 times as much as those at State College, while zinc samples at Kearny, New Jersey, corroded about 2.6 times as much (the concentration was 131 $\mu g/m^3$ at Elizabeth, New Jersey, near Kearney).

It should be concluded, however, that the variation in corrosion rates referred to above are due only to differences in particulate matter, since there are significant differences in gaseous pollutant concentrations between State College and the other areas. Even highly resistant metals have shown corrosion rates which are progressively larger over the following range of environments; (1) a rural semiarid site; (2) a rural farm enviornment; (3) an urban industrial area; (4) a heavy industrial area. In Chicago and St. Louis, steel panels were exposed at a number of sites, and measurements were taken of corrosion rates and of levels of sulfur dioxide and particulates. In St. Louis, except for one exceptionally polluted site, corrosion losses correlated well with sulfur dioxide levels, averaging 30 percent to 80 percent higher than corrosion losses measured in nonurban locations. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO_2 level of 0.03 ppm). Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Although suspended particulate levels measured in Chicago with high-volume samplers correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations had the dominant influence on corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 percent to 17 percent weight loss in steel panels) at annual average sulfur dioxide concentrations in the range of 0.03 ppm to 0.12 ppm, and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Particles play a significant role in corrosion and damage to electronic equipment of all kinds, even when precious metals are used to minimize such effects. The contacts in electrical switches are vulnerable to chemical or mechanical action by particulates. The particulates commonly found in industrial and urban atmospheres are serious factors in the corrosion and failure of electrical connectors and circuits.

The ability of particulates to damage and soil buildings, sculpture, and other structures is particularly great in cities where large quantities of coal and sulfur-bearing fuel oil are burned. Particles may cause deterioration to many types of masonry by acting as reservoirs for the harmful acids generated by the combustion of these fuels. In addition to direct erosion and physical degradation of materials, particles stick to surfaces with which they come in contact, forming a film of tarry soot and grit which may or may not be removed by the action of rain. The result is a dingy, soiled appearance, and much money and effort must be spent to sandblast off the sooty layers that accumulate.

Particles also may soil the painted surfaces of walls, ceilings, and the exteriors of homes and buildings, and, under certain circumstances, may cause them to become stained and pitted. Automobile finishes, for example, have been damaged by particulate matter emitted from nearby industrial operations. Water soluble chlorides and sulfates—mostly the iron, copper, calcium, and zinc salts—are commonly found in particulate samples from cities and in rainwater and may cause blistering and enhance the weathering of painted surfaces. Particles may settle on painted surfaces before the paint has dried, thus producing imperfections and reducing the ability of the paint to protect the surface. Such surfaces are likely to soon require refinishing.

The soiling of clothing, curtains, and similar textiles makes them unattractive and diminishes their use. The extent of soiling is influenced by a number of factors, including the temperature, the relative humidity, and the wind conditions. Small particles may penetrate deep into the fibers of curtains hanging in open windows. Curtains weakened by such exposure to atmospheric soiling and acidity deteriorate in a characteristic manner. The vulnerability of textile products to the acid components of airborne particles also depends on the composition of the material. Cellulosic fibers, for example, such as cotton, linen, hemp, jute, and man-made rayon, are particularly sensitive to attack from such substances as sulfurous and sulfuric acids. In addition to the aesthetic degradation and the nuisance created by particulate matter, direct costs may be associated with the increased rate of deterioration of textiles, the extra cleaning required, and the manufacturing expenses for fabrics that are more resistant to air pollution.

G. REFERENCES

1. Sheleikhovskii, G. V. "Smoke Pollution of Towns." Akademiya Kommunal'nogo Khzyaistva im. K. D. Pamfilova. Academy of Municipal Economy im. K. D. Pamfilova. Izdatel'stvo Ministerstva Kommunal'nogo Khozyaistva RSFSR, Moskva-Leningrad, 1949. (Translated from Russian and Published for the National Science Foundation, Washington, D.C. by the Israel Program for Scientific Translations, Jerusalem, 1961.)
2. Greenburg, L. and Jacobs, M. B. "Corrosion Aspects of Air Pollution." Am. Paint J., Vol. 39, pp. 64–78, 1955.
3. Vernon, W. H. J. "The Corrosion of Metals." J. Roy. Soc. Arts, Vol. 97, pp. 578–610, 1949.
4. Vernon, W. H. J. "A Laboratory Study of the Atmospheric Corrosion of Metals." (Parts 2 and 3.) Trans. Faraday Soc., Vol. 31, pp. 1668–1700. 1935.
5. Preston, R. St. J. and Sanyal, B. "Atmospheric Corrosion by Nuclei." J. Appl. Chem., Vol. 6, pp. 28–44, 1956.
6. Tice, E. A. "Effects of Air Pollution on the Atmospheric Corrosion Behavior of Some Metals and Alloys." J. Air Pollution Control Assoc., Vol. 12, pp. 533–559, 1962.

Hudson, J. D. "Present Position of the Corrosion Committee's Field Tests on Atmospheric Corrosion (Unpainted Specimens)." J. Iron Steel Inst., Vol. 148, pp. 161–215, 1943.

8. Larrabee, C. P. and Ellis, O. B. "Report of a Subgroup of Subcommittee VII on Corrosiveness of Various Atmospheric Test Sites as Measured by Specimens of Steel and Zinc. Committee B-3, American Society for Testing Materials." Am. Soc. Testing Mater. Proc., Vol. 59, pp. 183–201, 1959.
9. Copson, H. R. "Report of Subcommittee VI. Committee B-3, American Society for Testing Materials." In: Corrosion of Nonferrous Metals Symposium, Am. Soc. Testing Mater., Spec. Tech. Pub. 175, 1955.
10. "Air Pollution Measurements of the National Air Sampling Network (Analysis of Suspended Particulates, 1957–1961)." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Washington, D.C., 1962.
11. Upham, J. B. "Atmospheric Corrosion Studies in Two Metropolitan Areas." J. Air Pollution Control Assoc., Vol. 17, pp. 398–402, 1967.
12. Larrabee, C. P. "Effect of Composition and Environment on Corrosion of Iron and Steel." Am. Soc. Metals, pp. 30–50, 1956.
13. Antler, M. and Gilbert, J. "The Effects of Air Pollution on Electric Contacts." J. Air Pollution Control Assoc., Vol. 13, pp. 405–415, 1963.
14. Williamson, J. B. P., Greenwood, J. A. and, Harris, J. "The Influence of Dust Particles on the Contact of Solids." Proc. Roy. Soc., Ser. A (London), Vol. 237, pp. 560–573, 1956.
15. Carey, W. F. "Atmospheric Deposits in Britain: A Study in Dinginess." Intern. J. Air Pollution, Vol. 2, pp. 1–26, 1956.
16. Meller, H. B. and Sisson, L. B. "Effects of Smoke on Building Materials." Ind. Eng. Chem., Vol. 27, pp. 1309–1312, 1935.
17. Le Clerc, E. "Economic and Social Aspects of Air Pollution." In: Air Pollution (World Health Organization), Columbia University Press, New York, 1961, pp. 279–291.
18. Yocom, J. E. "The Deterioration of Materials in Polluted Atmospheres." J. Air Pollution Control Assoc., Vol. 8, pp. 203–208, 1958.
19. Cartwright, J. Nagelschmidt, G., and Skidmore, J. W. "The Study of Air Pollution with the Electron Miscroscope." Quart. J. Roy. Meteorol. Soc., Vol. 82, pp. 82–86, 1956.

20. Giles, C. H. "Adsorption. II. In Air Pollution and in Natural Processes." Chem. Ind. (London), Vol. 19, pp. 770–781, 1964.
21. Holbrow, G. L. "Atmospheric Pollution: Its Measurement and Some Effects on Paint." J. Oil Colour Chemists Assoc., Vol. 45, pp. 701–718, 1962.
22. Fochtman, E. C. and Langer, G. "Automobile Paint Damaged by Airborne Iron Particles." J. Air Pollution Control Assoc., Vol. 6, pp. 243–247, 1957.
23. Tabor, E. C. and Warren, W. V. "Distribution of Certain Metals in the Atmosphere of Some American Cities." Arch. Ind. Health, Vol. 17, pp. 145–151, 1958.
24. Waller R. E., Brooke, A. G. F., and Cartwright, J. "An Electron Miscroscope Study of Particles in Town Air." Intern. J. Air Water Pollution, Vol. 7, pp. 779–786, 1963.
25. Graff-Baker, C. "The Effect of Dust Particles on the Electrical Resistance and Anti-Corrosive Properties of Varnish and Paint Films." J. Appl. Chem., Vol. 8, pp. 500–598, 1958.
26. Parker, A. "The Destructive Effects of Air Pollution on Materials." The Sixth Des Voeux Memorial Lecture. (Presented at the Annual Conference of the National Smoke Abatement Society, Bournemouth, England, September 28, 1955.)
27. Rees, W. H. "Atmospheric Pollution and Soiling of Textile Materials." Brit. J. Appl. Phys., Vol. 9, pp. 301–305, 1958.
28. Petrie, T. C. "Smoke and the Curtains." Smokeless Air, Vol. 18, pp. 62–64, 1948.
29. Tebbens, B. D. "Residual Pollution Products in the Atmosphere." In: Air Pollution, Chapter 2, Vol. 1, 1st edition, A. C. Stern (ed.), Academic Press, New York, 1962, pp. 23–40.

Chapter 5

ECONOMIC EFFECTS OF ATMOSPHERIC PARTICULATE MATTER

Table of Contents

Page
A. INTRODUCTION 79
1. The Role of Economic Analysis 79
2. Gross Estimates 80
B. EARLY ECONOMIC STUDIES 81
1. Pittsburgh 81
2. Other Early Economic Studies 81
C. RECENT EFFORTS 81
1. Household Effects 81
2. Property Value Studies 83
3. Productivity Studies 84
D. SUMMARY 84
E. REFERENCES 85

List of Figures

Figure
5–1 Maintenance Frequency as a Function of Particle Concentration in the Upper Ohio River Valley and the National Capital Area (for Households of Above-average Income) 82

List of Tables

Table
5–1 Estimated Costs Due to Air Pollution (Mellon Institute Pittsburgh Study) 81
5–2 Differences in Cleaning Costs Incurred at Steubenville and at Uniontown 82

Chapter 5

ECONOMIC EFFECTS OF ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

1. The Role of Economic Analysis

It is, perhaps, misleading to cordon off the so-called economic effects of particulate pollutants from the effects which are the grist of this document. All of the effects discussed in this document have an economic dimension. Some of the economic dimensions are easier to measure than others. It is difficult to determine, for example, what is the value of preserving the health, or indeed the life, of individuals adversely affected by pollution. It is somewhat easier to discover differences in expenditure patterns attributable to the presence of air pollution.

Difficult or easy, such valuation is a logically necessary part of the air quality standards decision process. Communities desirous of a more favorable environment are confronted with the need for two bodies of fact. First, there are the physical and technological laws that govern the generation, transport, and control of air pollution. Second, there are the undesirable effects of pollution on man and his environment. Furthermore these two realities conspire to produce a dilemma. On the one hand, if the undesirable effects of air pollution are to be avoided, economic resources that might otherwise be used to satisfy other worthy community objectives will have to be used for air pollution control instead, and the other objectives foregone. If, on the other hand, all these other objectives are retained undiminished, the effects of a polluted environment must be endured. It is evident that some sort of balance must be struck. Precisely where this balance is achieved is a matter for the individual community to decide.

The tools of economic analysis can illuminate the nature of this dilemma and can even, with some qualification, indicate a way out. It is sometimes suggested, for instance, that the monetary value of the undesirable consequences of *not* controlling air pollution be balanced against the consequences of instituting control. This is the thrust of the much-heralded cost-benefit analysis, under which the decision maker must consider the available alternatives and choose that which seems most favorable. Comparisons are made insofar as possible in monetary terms, with the dollar serving as a common denominator. It is the purpose of this chapter to review the as-yet-small body of literature on the economic value of the effects listed throughout this volume.

A major difficulty in assessing damages due to particulate air pollution lies in isolating the effects of particulates from those of other classes of air pollutants, such as sulfur oxides, ozone, oxides of nitrogen, and others, including odors. The present state of the arts with respect to the measurement transport, ambient dosages, and the short-term and long-term effects of the several air pollutants in various concentrations, durations, and conditions, demands that increased attention, including an expanded research effort, be given to the determination of the effects of air pollution and the economic costs of those effects.

It is a major objective of research into the economic effects of air pollution to establish quantitative relationships between varying levels of pollution and outcomes (the effects of those levels) and ultimately to arrive at acceptable measures of the economic costs attributable to them. In turn, when such costs are averted they become the benefits of air pollution control.

2. Gross Estimates

A review of the physical effects cited in previous chapters suggests the possibility of estimating damage factors and costs of damages. The basic measurement procedure involves four steps:

1. Identification of nonoverlapping categories of air pollution damage;
2. An estimate of the total value of the category, regardless of the air pollution effects;
3. Determination of an air pollution damage factor incorporating assumptions or knowledge of the relationships between the total size of the category and the damages due to air pollution; and
4. Application of this damage factor to the total value of the category, in order to estimate the damages due to air pollution and those due to the particular pollutant.

For example, one category of effects is the corrosion of steel structures which necessitates painting. An estimate might be developed along the following lines. The Steel Structures Painting Council, Pittsburgh, Pa., estimated the annual cost of corrosion-inhibiting coatings applied to steel structures at $500 million in 1965.[1]* This figure is adjusted at an annual rate of increase of 6 percent, yielding a 1968 estimate of $590 million.[2] The Council estimated that a large proportion of the paint was applied because of factors such as humidity and chemicals. They suggested that particulate air pollution may be responsible for about 5 percent of the need for corrosion-inhibiting paints. This factor was used to determine damages due to air pollution and the cost of the paint, estimated at $29.5 million. Added to this is the labor required to apply the paint. Labor is approximately 2.5 times the cost of the paint as shown in the 1949 study by Uhlig[3]** The total paint and labor gives a total estimate of $103 million per year as the cost of painting steel structures because of air pollution.

Commercial laundering, cleaning, and dyeing is another category of loss due to dirty air. In 1963, commercial laundering, cleaning, and dyeing costs amounted to $3,475 million.[4] Between 1958 and 1963, these costs were increasing by 5 percent annually. The adjusted figure for this category for 1968 thus becomes $4,350 million.

The Beaver Report found evidence suggesting that one-third of laundry costs in England were attributable to the effects of air pollution.[6] A damage factor for this category for the United States for the cost of commercial laundering, cleaning, and dyeing due to air pollution becomes $870 million.

Transportation delays is another good example of an economic loss. Air pollution is a major cause of reduced visibility. The Civil Aeronautics Board reported that in 1962 low visibility resulting from smoke, haze, dust, and sand in the air possibly caused 15 to 20 plane crashes.[7] Other costs of low visibility include travel delays, diversions, cancellations, and the cost of transportation to individuals who wish to escape polluted areas on weekend trips. Land transportation costs may well include similar losses. In air travel alone, the total costs of diversion, cancellation delays, and crashes were estimated at $803 million for 1960.[8] Today, the cost would be higher. If as little as 5 to 10 percent is attributed to air pollution, $40 to $80 million or more is involved.

Automobile washing is another example. Expenditures on automobile washing amounted to $143 million in 1963.[9] This category grew by almost 10 percent per year between 1958 and 1963. Extending this rate yields an estimate of $210 million for car washing in 1968. The $210 million is increased by another 50 percent to $300 million also, to adjust for the fact that washing by the car owner is not included in the lower figure.

The largest proportion of automobile washing is probably caused by particulate air pollution. A damage factor of 80 percent is assumed for car washing, and expenditures for automobile washing due to the effects of air pollution would be about $240 million for 1968.

---

* Estimate supplied on March 11, 1965; contacted again on April 24, 1968, but no further work had been done.

** This 2.5 labor factor still holds true, as can be seen by comparing total recorded household repair expenditures versus expenditures for materials in recent years. In 1963, for example, the ratio was 2.3.

If this procedure could be repeated for nonoverlapping categories of damage due to particulate air pollution, additional estimates of such damages could be developed.

B. EARLY ECONOMIC STUDIES

1. Pittsburgh

One of the first comprehensive surveys of damage due to air pollution in the United States was conducted in Pittsburgh during the years 1912 to 1913 by the Mellon Institute. The estimates in the Pittsburgh investigation were based on economic losses due to dustfall and smoke and were obtained through interviews of individuals. The items of cost and the total cost to the community were ascertained as shown in Table 5-1.

The Mellon Institute investigators arrived at an annual per capita cost of $20 in 1913. Health effects were not included; aesthetic losses at the time were judged to be $5 per person above the $20.[10]

Table 5-1.—ESTIMATED COSTS DUE TO AIR POLLUTION (MELLON INSTITUTE PITTSBURGH STUDY.[10]

Causes of expenditure	*Cost*
To smoke maker:	
Imperfect combustion	$1,520,740
To individual:	
Laundry bills	1,500,000
Dry cleaning bills	750,000
To household:	
Exterior painting	330,000
Sheet metal work	1,008,000
Cleaning and renewing wall paper	550,000
Cleaning and renewing lace curtains	360,000
Artificial lighting	84,000
To wholesale and retail stores:	
Merchandise	1,650,000
Extra precautions	450,000
Cleaning	750,000
Artificial lighting	650,000
Department stores	175,000
To quasi-public buildings:	
Office buildings	90,000
Hotels	22,000
Hospitals	55,000
Total	$9,944,740

2. Other Early Economic Studies

A comprehensive study of the economic effects of air pollution in Great Britain is described in the Beaver Report in 1954.[6] This study considered both direct costs and efficiency losses. The direct costs included laundry and domestic cleaning; the cleaning, painting, and repair of buildings; the corrosion of metals and consequent cost of replacement and of providing protective coverings; damage to goods; additional lighting; and extra hospital and medical services. Efficiency losses were represented by the effects on agriculture of damage to soil, crops, and domestic animals; interference with transport; and reduced human efficiency due to illness.

Le Clerc, writing in 1961 about economic losses due to air pollution, cited data from France and Great Britain, as well as some general data on economic losses in the United States. The foreign data were quoted in local monetary units, which have varied over the years in relation to the value of the dollar. Even those figures relating to the United States are cited in the context of the value of the dollar at the time of study. Some of the data contained estimates on medical services; others did not.[11]

In these two studies no attempt was made to relate economic losses to the ambient particle concentration.

C. RECENT EFFORTS

1. Household Effects

More recent attempts to assess specific economic losses due to air pollution have been made by Michelson and Tourin in the Upper Ohio River Valley and elsewhere.[12-16] The investigators made a comparative analysis of Steubenville, Ohio and Uniontown, Pennsylvania. The socioeconomic and climatic data were generally comparable, whereas the air pollution levels, using particle concentration ($\mu g/m^3$) as an index, were dissimilar. Uniontown had an annual average of 115 $\mu g/m^3$, while Steubenville had an annual average of 235 $\mu g/m^3$.

Six categories of possible loss were studied in each community:

1. Outside maintenance frequencies of houses (cleaning painting, etc.);

2. Inside maintenance frequencies of houses and apartments (walls, windows, drapes, curtains, venetian blinds, carpets, and furniture);
3. Laundering and dry cleaning of clothing (with distinction between summer and winter maintenance practices);
4. Maintenance of women's hair and facial care;
5. Inside maintenance of offices (cleaning and painting); and
6. Store operation and maintenance (cleaning, painting, and other maintenance items; losses due to spoilage of merchandise).

Data from only the first four categories were used in the comparative analysis owing to the heterogeneity of the establishments interviewed and the small number of respondents for the last two categories, nos. 5 and 6. It will be noted that these items of cost are related for the most part to the effects of particulate matter rather than to all air pollutants.

For each loss category three types of data were sought:

1. Activity frequency;
2. Incidence (i.e., the proportion of the population to which various frequencies were applicable); and
3. Socioeconomic characteristics (i.e., household income, educational level).

Questionnaires were designed separately for each area of activity to collect accurate information on frequency of maintenance operations. Although income data were obtained in steps of $2000, only two income categories were used in the final comparative analysis: "less than $8000" and "over $8000." The maintenance frequency factor was calculated, and the frequency factors were converted into dollar values.

Table 5–2 shows the calculated extra *per capita* and total costs incurred by Steubenville as a result of air pollution.

The Upper Ohio River study also included a third city, Martins Ferry, Ohio, where the particle concentrations were roughly midway between those of the first two cities. The average frequency of maintenance operations in Martins Ferry fell almost precisely midway between those in Steubenville and Uniontown.

The curve relating costs of air pollution and suspended particle concentrations, the latter being used as an index of air pollution, was found by these investigators to be essentially a straight line. Using these data, the authors extrapolated the straight line of Figure 5–1 back to the average particle concentration of the rural stations of the National Air Sampling Network (NASN).

Table 5–2.—DIFFERENCES IN CLEANING COSTS INCURRED AT STEUBENVILLE AND AT UNIONTOWN.[12]

Activity	Gross cost Differences (Steubenville over Uniontown) Annual	Per Capita*
Outside maintenance of houses	$ 640,000	$17
Inside maintenance of houses and apartments	1,190,000	32
Laundry and drycleaning	900,000	25
Hair and facial care	370,000	10
Total	$3,100,000	$84

* Based on estimated 1959 population of 36,400.

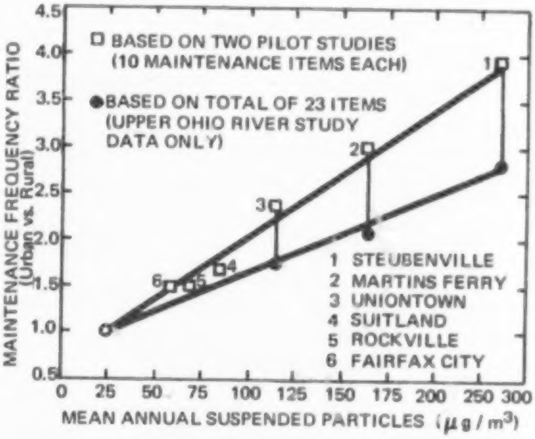

FIGURE 5–1. Maintenance Frequency as a Function of Particle Concentration in the Upper Ohio River Valley and the National Capital Area (for Households of Above-average Income). (This figure shows that maintenance frequency increases almost linearly with the mean suspended particle concentrations.)

The mean annual rural station concentration of suspended particulate matter for 1959 to 1961 was 25 μ/m^3.

A similar investigation of comparative costs due to air pollution was conducted by Michelson and Tourin in the metropolitan Washington, D.C., area in 1967. The character of metropolitan Washington differs markedly from that of Steubenville, the latter having much heavy industry (steel mills, etc.). The Washington pilot study was based on a selection of families in private dwellings only.

Four communities of the Washington, D.C. area (Rockville Maryland; Suitland, Maryland; Hyattsville, Maryland; and Fairfax City, Virginia) were originally selected for study because they represented extremes in particle concentrations for outlying areas. At the time, they all had a large proportion of middle-income families. The returned questionnaires were processed (according to the replies) by income group. Since it was expected that families in the very low and very high income brackets would be relatively insensitive to particulate pollution, only the data in the $10,000-to-$14,000 bracket were utilized. Deficiencies in the air quality data for Hyattsville precluded its inclusion in the analysis.

Results from the three suburban Washington communities compared quite favorably with the results from the Upper Ohio River Valley study on the ten items which were common to both studies. Figure 5–1 shows the relationship between suspended particle concentrations and maintenance frequency ratio (urban/rural) for the two studies combined, as well as for the Upper Ohio River Valley study alone.

It may be noted that in both sets of data the association between the maintenance frequency ratios and suspended particulates is taken to be linear over the range of the actual data, rather than an expected leveling out of the "curve" at extremely high concontrations of particulates. Maintenance frequency ratios were not translated into costs in the suburban Washington communities because of widely different socioeconomic and time differences between the two study areas. The importance of the studies lies not so much in the estimates of cost, which are likely to vary in place and time, but in the estimates of differences in maintenance. It should be noted that the use of common cost/maintenance operation has the effect of understating the true difference in expenditures for the items studied between the two areas.

The Michelson-Tourin studies have suggested one approach to the problem of estimating the costs of some of the effects of particulate air pollution. The results of investigations to date are highly suggestive of the existence of a significant relationship between the costs of household and personal maintenance and the existence of air pollution.

A word of caution in the interpretation of these results is in order. It is well known that causation and statistical correlation are not one and the same. There are a number of other factors, not investigated in the studies of Michelson and Tourin, which could be highly correlated with air pollution, and which could be casually related to the frequency with which the various personal hygiene and property maintenance routine studied are performed. For example, highly urbanized areas are likely to have both relatively high levels of air pollution and a relative abundance of the materials and skills necessary for the performance of hygiene and maintenance operations (e.g., drycleaning establishments, beauticians, etc.). More work is needed to find out unambiguously just what forces are operating.

2. Property Value Studies

In 1967 the results of investigations into the effect of air pollution on property and other values in St. Louis, Syracuse, and Philadelphia were published in a book by Dr. Ronald Ridker entitled *Economic Costs of Air Pollution—Studies in Measurement.*[17] The studies were somewhat inconclusive; they dealt generally with air pollution effects and did not attempt to isolate the effects of particulates. Nevertheless, the effort represents an important methodological milestone because it describes some important attempts to determine the economic extent of air pollution damage and includes some practical suggestions for the guidance of investigators. For example, in the chapter on "Soiling and Material—Damage Studies," Dr. Ridker expresses pessimism with respect to the value of additional studies based on analysis of currently available data and, citing the high cost of surveys, proposes as an alternative "a special type of experimental approach to the gathering of the relevant economic information."

The recommended approach would seek to establish the physical and biological effects of various types and combinations of air pollution in varying concentrations and durations. It would include study of the effects of weather conditions and attempt to establish predictive relationships that could describe the amount of damage that would result from specific exposure conditions. The resulting damage functions could then be subjected to economic analysis in an effort to translate effects into costs.

Ridker's studies of air pollution and property values were limited to cross section studies. They tended to show an inverse relationship between property values and air pollution levels as measured by mean values for sulfation rates.

Other investigators, including T. D. Crocker,[18] have demonstrated that the difference in land value between a polluted and a nonpolluted area is an appropriate measure of potential gain from an abatement policy.

The results of property-value studies appear encouraging in that they are a step in the direction of defining a function relationship between reductions in air quality and economic loss. They supplement the studies of effects of air pollution described previously.

3. Productivity Studies

Many feel and some are convinced that air pollution, and especially the combination of suspended particulates with sulfur dioxide, is associated with an increasing incidence of lung and respiratory ailments and heart disease.[19, 20]

To the extent that particulate air pollution affects the respiratory tract and produces or contributes to illness among the working population it may substantially reduce human productivity and result in economic losses. Economic losses would result from work-loss days, reduced worker productivity, and a shortened productive work life. At this time, it is not possible to do more than speculate about the possible magnitude of this possibly significant economic loss.

Efforts to provide useful economic estimates of the effects of disease have long been a major problem for economists. Nevertheless, estimates have been made for a number of diseases and agreement appears to be developing on measurement methods. The most economically useful measure of the effects of reduced productivity would be the capitalized value or the present discounted value of gross lost production.[21, 22] Progress is reported in methods of valuing a human life, apart from livelihood. All efforts to construct acceptable estimates of the costs of poor health or early death attributable to air pollution depend on the results of research that can establish dependable cause and effect relationships.

D. SUMMARY

The cost of painting steel structures because of air pollution, particularly due to particulate matter, has been estimated at about $100 million a year. The annual cost of commercial laundering, cleaning, and dyeing due to air pollution is estimated at $850 million. The adverse effects of air pollution on air travel were estimated minimally at $40–$80 million in 1960; obviously, they would be greater today. Expenditures for car washing, including washing by the car-owner involved, due to air pollution are estimated at about $250 million in 1968. This assumes the principal cause of frequent car washing is particulate air pollution. The Michelson and Tourin studies, despite their stated deficiencies, reveal a general relationship between levels of particulate air pollution and increased frequency of household maintenance operations. Unfortunately, it is not readily possible to convert these relationships into cost relationships that would be uniformly applicable to all communities.

Studies of air pollution and residential property values, while limited by data availability, strongly suggest that a statistically significant inverse relationship may exist between higher levels of particulate and other air pollution and residential property values.

Efforts to provide useful economic information about the effects of air pollution (particulates) on human health and productivity losses are incomplete because they depend on results of other research efforts aimed at the establishment of dependable cause and effects relationships.

It may be possible to develop acceptable gross estimates of air pollution damage based on analysis within a group of non-overlapping damage categories as suggested in the introduction to this chapter. If such efforts are carried out with care, they may provide useful guides to policy determination. Considerably more attention needs to be given to the physical and biological effects of air pollution before definitive estimates of the economic costs of air pollution effects can be made.

Such estimates are not the final step, however. It will then be necessary to determine the costs averted (or benefits) by a particular mechanism or program of air pollution control, in relation to the costs of control.

E. REFERENCES

1. Steel Structures Painting Council, Pittsburgh, Pa., Correspondence dated March 11, 1965.
2. "Statistical Abstract of the United States: 1967." 88th edition, U.S. Bureau of the Census, Washington, D.C., 1967, Table 1067.
3. Uhlig, Herbert H., "The Cost of Corrosion to the United States." Chem. Eng. News, Vol. 27, pp. 2764–2767, 1949.
4. "Statistical Abstract of the United States, 1967." 88th edition, U.S. Bureau of the Census, Table 1193.
5. "U.S. Census of Business: 1963, Selected Services." Vol. 6, U.S. Dept. of Commerce, Washington, D.C., 1963, Table I.
6. "Committee on Air Pollution Report, A Report to the Minister of Housing and Local Government, Sir Hugh Beaver, Chairman." Her Majesty's Stationery Office, London, 1954.
7. "The Polluted Air We Breathe." American Federationist, Vol. 71(2), pp. 6–11, 1964.
8. Fromm, Gary, "Civil Aviation Expenditures," In: Measuring Benefits of Government Investments, The Brookings Institution, Washington, D.C., 1965, Table 2.
9. "U.S. Census of Business: 1963, Selected Services Area Statistics," Vol. 7, U.S. Dept. of Commerce, Washington, D.C., 1963, Table 1–8.
10. O'Connor, J. J., Jr. "The Economic Cost of the Smoke Nuisance to Pittsburgh." University of Pittsburgh, Mellon Institute of Industrial Research and School of Specific Industries, Pittsburgh, Pa., Smoke Investigation Bulletin 4, 1913.
11. Le Clerc, E. "Economic and Social Aspects of Air Pollution." In: Air Pollution (World Health Organization), Columbia University Press, New York, 1961, pp. 279–291
12. Michelson, I. and Tourin, B. "Comparative Method for Studying Cost of Air Pollution." Public Health Rept. Vol. 81(6), pp. 505–511, June 1966.
13. Michelson, I. and Tourin, B., "Report on Study of Validity of Extension of Economic Effects of Air Pollution Data from Upper Ohio River Valley to the Washington, D.C. Area." Public Health Service Contract PH 27–68–22, November 8, 1967.
14. Michelson, I. and Tourin, B., "Household Cost of Living in Polluted Air Versus the Cost of Controlling Air Pollution in the Twin Kansas Cities Metropolitan Area." Public Health Service Contract PH27–68–21.
15. Michelson, I. and Tourin, B., "Household Cost of Living in Polluted Air in the Washington Metropolitan Area." Washington, D.C. Metropolitan Area Air Pollution Abatement Conference, January 1968.
16. Huey, N. "Economic Benefit from Air Pollution Control." Preprint. (Presented at the New York-New Jersey Metropolitan Area Air Pollution Abatement Activity, February 5, 1968.)
17. Ridker, R. G., "Economic Costs of Air Pollution: Studies in Measurement." Frederick A. Praeger, New York, 1967.
18. Crocker, T. D., "Some Economic Aspects of Air Pollution Control with Special Reference to Polk County, Florida." Research Report to U.S. Public Health Service, Grant AP–00389.
19. Committee on Pollution, National Research Council, "Waste Management and Control." A Report to the Federal Council for Science and Technology, National Academy of Science, National Research Council, Washington, D.C., Pub. 1400, 1966.
20. "Restoring the Quality of Our Environment." Environmental Pollution Panel, President's Science Advisory Committee, The White House, November 1965.
21. Weisbrod, B. A., "Economics of Public Health." University of Pennsylvania Press, Philadelphia, 1961.
22. Klarman, H. E., "Syphillis Control Programs." In: Measuring the Benefits of Government Investments, R. Dorfman (ed.), The Brookings Institution, Washington, D.C., 1965.

Chapter 6

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VEGETATION

Table of Contents

Page

A. INTRODUCTION ... 89

B. EFFECTS OF SPECIFIC DUST ON VEGETATION ... 89
- 1. Cement-Kiln Dust ... 89
  - a. Direct Effects ... 89
    - (1) Nature of Dust Deposition ... 89
    - (2) Range of Effects ... 91
    - (3) Dust Components Involved ... 92
  - b. Indirect Effects ... 93
- 2. Flourides ... 93
- 3. Soot ... 94
- 4. Magnesium Oxide ... 94
- 5. Iron Oxide ... 94
- 6. Foundry Dusts ... 94
- 7. Sulfuric Acid Aerosols ... 94

C. EFFECTS OF DUSTS ON ANIMALS BY INGESTION OF VEGETATION ... 95

D. SUMMARY ... 95

E. REFERENCES ... 96

List of Figures

Figure

6–1 Bean Plants Dusted with Cement-Kiln Particles ... 90

6–2 Cement-Kiln Dust on Fir Tree Branches ... 91

6–3 Cement-Kiln Dust on Fir Tree Branches ... 91

Chapter 6

EFFECTS OF ATMOSPHERIC PARTICULATE MATTER ON VEGETATION

A. INTRODUCTION

Little is known about the effects of particulate matter on vegetation and little research has been done on the subject. There has been far more research on gaseous pollutants, many of which are readily recognized as serious toxicants to a variety of plants. Published experimental results, mostly from Germany, are confined principally to settleable dusts emitted from the kilns of cement plants. There are a few reports on effects of fluoride dusts, soot and particulate matter from certain types of metal processing. Sulfuric acid aerosols have also been studied in Los Angeles, where deposition of acid droplets has injured the leaves of vegetation. Most of the research was related to the direct effects of dusts on leaves, twigs, and flowers as opposed to indirect effects from dust accumulation in the soil. Because of the dearth of experimental results, the tenor of many reports is directed as much to the question, "Do dusts in fact have deleterious effects on plants?" as to the question of extent of injury. It is thus reasonable to anticipate some disagreement. Such information as there is refers to specific dusts rather than to the conglomerate that is usually measured as urban or rural dustfall (Chapter 1). The various specific pollutant dusts and their injurious effects on vegetation are discussed in the following sections.

B. EFFECTS OF SPECIFIC DUSTS ON VEGETATION

1. Cement-Kiln Dust

Cement-kiln dust is the dust contained in waste gases from the kilns and is not derived directly from processing of cement. It is apparent from some reports, however, that the composition of wastes from different kilns operating at different efficiencies varies considerably, and at times the effluents may contain cementitious materials that more properly belong in the finished product. Another important factor to consider is that literature reports describing effects of dust deposited on various plants in the field relate to kiln-stack materials, whereas experimental dusts applied in laboratory or field studies were taken from various collectors in the waste-gas system between the kiln and the stack. Differences in results that may be due to this factor have not been reconciled.

a. Direct Effects

(1) *Nature of Dust Deposition.*—Most of the reports concerning harmful effects of cement-kiln dust on plants stress the fact that crusts form on leaves, twigs, and flowers. As early as 1909–1910 Peirce[1] and Parish[2] noted in California that settled dust in combination with mist or light rain formed a relatively thick crust on upper leaf surfaces of affected plants. The crust would not wash off and could be removed only with force. The central theme about which Czaja[3–6] builds his case for harmful effects is the crust formation in the presence of free moisture. He states that crust is formed because some portion of the settling dust consists of the calcium silicates which are typical of the clinker (burned limestone) from which cement is made. When this dust is hydrated on the leaf surface, a gelatinous calcium silicate hydrate is formed, which later crystallizes and solidifies to a hard crust. When the crust is removed, a replica of the leaf surface is often found, indicating intimate contact of dust with the leaf. The relatively thick crust formed from continuous deposition is confined to the upper leaf surface of deciduous species but completely encloses needles of

conifers. Prolonged dry periods during the time dust is deposited provide no opportunity for hydration, and crusts are not formed. Dust deposits which are not crusted are readily removed by wind or hard rain.

Darley [3] applied kiln dusts of particle size less than 10μ at rates of 0.05 mg/cm^2-day to 0.38 mg/cm^2-day to leaves for two to three days in the laboratory. Water mist was applied several times each day. Even though the dust adhered to the leaf in a uniform layer, it did not appear to be crust-like, probably because the experiments were of short duration. Reduction in CO_2 uptake was reported in these experiments.

Leaves of bean plants dusted for two days with cement-kiln dust of 8μ to 20μ size at the rate of 0.47 mg/cm^2-day and then exposed to naturally occurring dew were moderately damaged.[4] The injury (Figure 6–1) appeared as a rolling of the margins of the leaves, and some interveinal tissues were killed. Leaves which were dusted but kept dry were not injured.

Photographs taken recently in Germany by Darley (Figure 6–2 and Figure 6–3) show incrustations of cement-kiln dust on branches of fir trees. Atmospheric levels of dust in this area were probably in excess of 85 tons/mile^2-month (0.1 mg/cm^2-day). Incrustations built up on the older twigs (Figure 6–2) and caused needles to fall prematurely. Some of the twigs were dead and incrustations were forming on the newest needles.

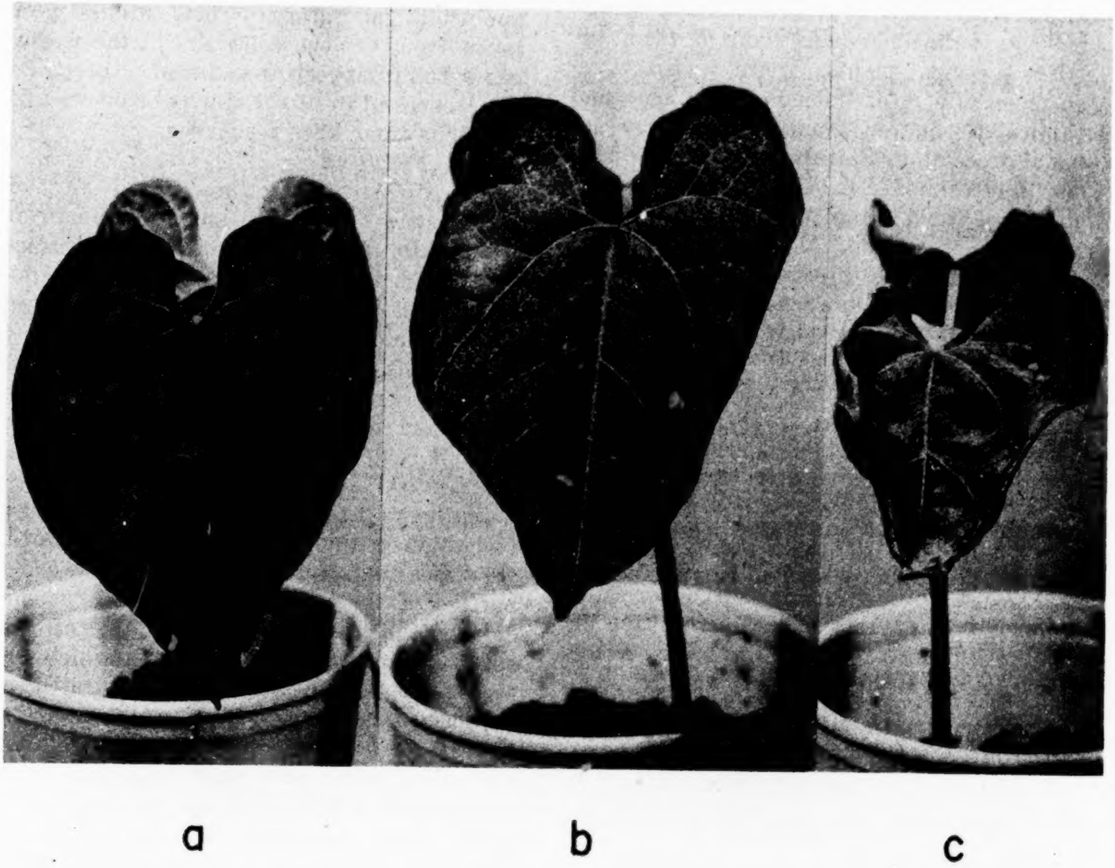

FIGURE 6–1. Bean Plants Dusted with Cement Kiln Particles. (a) Control Plant; (b) Dusted but Kept Damp; (c) Exposed to Natural Dew Formation After Each Day's Dusting.[4] (The photograph shows the effect of dusting bean plants for two days with particles (8μ to 20μ) from a cement kiln. The plant exposed to moisture (natural dew) and dust is most affected. The dusting rate was 0.47 mg/cm^2-day.)

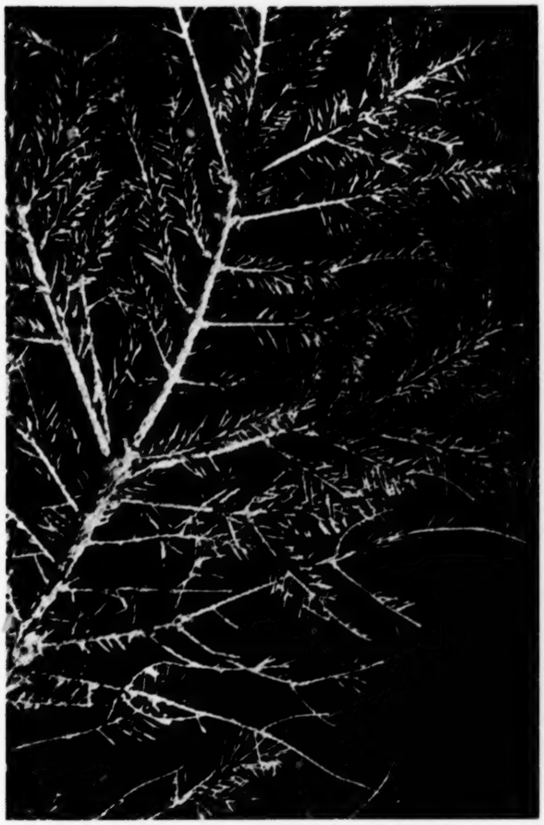

Figure 6–2. Cement-kiln Dust on Fir Tree Branches.[4] (Incrustation has built up on the older twigs of a fir tree exposed to a cement-kiln dustfall probably in excess of 1 mg/m^2-day. Needles have fallen prematurely.)

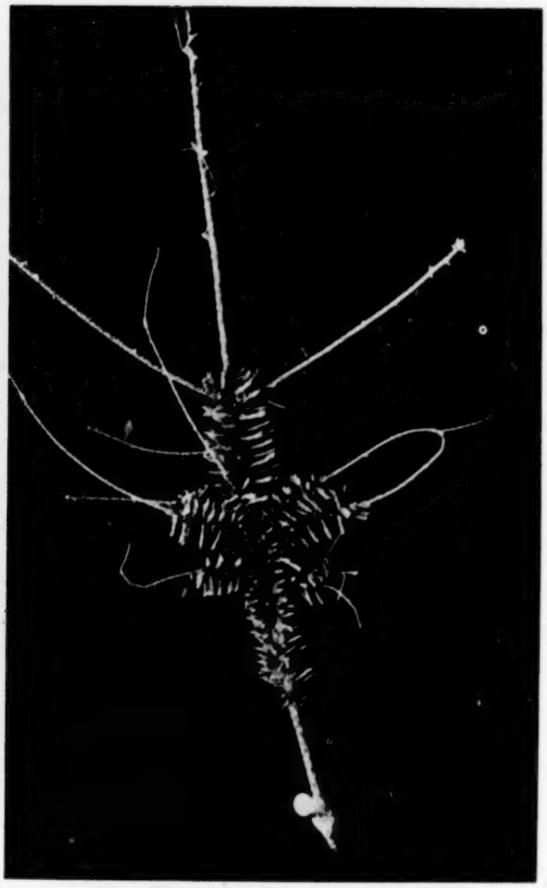

Figure 6–3. Cement-kiln Dust on Fir Tree Branches.[4] (Very heavy incrustation on a branch of a dead fir tree exposed to cement kiln dust (dustfall rates probably in excess of 0.1 mg/cm^2-day).)

The net effect was a shortening of each succeeding year's flush of growth. A dead tree had heavy incrustations on the branches (Figure 6–3).

(2) *Range of Effects.*—Peirce[1] demonstrated that incrustations of cement-kiln dust on citrus leaves interfered with light required for photosynthesis and reduced starch formation. This was later confirmed by Czaja[5] and Bohne[6] in a variety of plants. More recently, Steinhubel[7] compared starch reserve changes in undusted common holly leaves and those dusted with foundry dust. He concluded that the critical factor in starch formation was the light absorption by the dust layer, and that the influence on transpiration or over-heating of leaf tissue was of minor significance. Lecenier and Piquer (see Czaja[5]) attributed the reduced yields from dusted tomato and bean plants to interference with light imposed by the dust layer. Darley[3] demonstrated that dust deposited on bean leaves in the presence of free moisture interfered with the rate of carbon dioxide exchange, but no measurements of starch were made.

Czaja[5] stated that the hydration process of crust formation released calcium hydroxide. The hydrated crusts gave solutions of pH 10–12. Severe injury of naturally dusted leaves, including killing of palisade and parenchyma cells, was revealed by microscopic examination. The alkaline solutions penetrated stomata on the upper leaf surface, particularly the rows of exposed stomata on needles of conifer species, and injured the cells beneath. Czaja[5] stated that on broad-leaved species with stomata only on the lower leaf surface, the alkaline solutions first saponified the protective cuticle on the upper surface, permitting migration of the solution through the epidermis to the palisade and parenchyma tissues. Typical alkaline precipitation reaction with tannins, especially in leaves of rose and strawberry, was evidence that calcium hydroxide penetrated the leaf tissue. Bohne[6] described similar "corrosion" of tissues under the crust formed on oak leaves.

Czaja[7] has presented good histological evidence that stomata of conifers may be plugged by dust, preventing normal gas exchange by the leaf tissue. Uninhibited exchange of carbon dioxide and oxygen by leaf tissue is necessary for normal growth and development.

Bohne[6] reported a marked reduction of growth of poplar trees located about one mile from a cement plant after production in the plant was more than doubled. The change in growth rate was determined by the width of annual rings in the wood. Darley[8] observed a reduction of spring growth elongation on conifers in Germany, where the oldest needles were incrusted. He also noted that plants were stunted and had few leaves in the heavily dusted portions of an alfalfa field downwind from a cement plant in California. Plants appeared normal in another part of the field where there was no visible dust deposit. The dusted plants were also heavily infested with aphis and it was not clear whether the poor growth was due to the aphis feeding or a direct effect of the dust. Entomologists suggested that the primary effect of the dust may have been to eliminate aphid predators, thus encouraging high aphid populations, which in turn cause poor plant growth because of their feeding.

Anderson[9] observed in New York that cherry fruit set was reduced on the side of the tree nearest a cement plant. He demonstrated that the dust on the stigma prevented pollen germination. Schönbeck[10] treated a field planting of sugar beets biweekly with 2.5 g/m^2 of dust and observed that infection by leaf-spotting fungus, *Cercospora beticola*, was significantly greater than in nondusted plots. He postulated that the physiological balance was altered by dust increasing susceptibility to infection.

Pajenkamp[11] reviewed the unpublished work of several German investigators, some of whom had applied dust artificially to test plants, and stated that he was opposed to the view that dusts are harmful to plants. He concluded that depositions of from 0.75 g/m^2-day to 1.5 g/m^2-day (the latter amount representing the maximum that might be found in the vicinity of a cement factory) had no harmful effect on plants.

Raymond and Nussbaum[12] also stated that cement dusts have little effect on wild plants. On the other hand, Guderian[13] and Wentzel[14] disagreed with Pajenkamp and stated that the limited evidence at best presented a contradictory picture and that Pajenkamp had not cited Czaja's earlier work.[5, 7, 15, 16] They also pointed out that a deposit of 1.5 g/m^2-day was not maximum, since other workers had found up to 2.5 g/m^2-day, and Bohne[6] has since reported weekly averages of up to 3.8 g/m^2-day.

According to Czaja,[5] Ewert concluded that cement-kiln dust did not clog the stomata and that the crust might have a beneficial effect as protection against transpirational losses and a defense against fungi. Czaja also noted that the interpretation of evidence by Ewert is open to question because control plants were heavily infested with flea beetles, while test plants were not.

(3) *Dust Components Involved*—Detail on the injury to be expected from certain cement-kiln precipitator dusts was given by Czaja.[7] His work is based on comparisons of chemical composition of dusts and resultant injury to leaf cells of a sensitive moss plant, *Mnium punctatum*. A cut leaf was mounted in water on a microscope slide, and dust was placed in contact with the water at the edge of the cover slip. Any effect of the resultant solution on leaf cells could be observed directly. Eighteen of the dusts tested in this way fell into the following categories:

1. No permanent injury to living cells, but some plasmolysis from the con-

centration effect of the solution;

2. Slight injury to readily accessible cells, disruption of the cytoplasm, and displacement of chloroplasts; and
3. Severe injury to all cells of the leaflet.

Dusts were further described as follows: group 1, pH of 9.5–11.5, a relatively high rate of carbonation, and intermediate amount (19–29 percent) of clinker phase (calcium silicates), and characterized by a high (36–79 percent) amount of secondary salts; group 2, pH about 11, a high rate of carbonation, a lower (13–16 percent) clinker phase and characterized by a high (81–85 percent) proportion of raw feed; group 3, pH 11–12, a very slow carbonation rate and characterized by a high (17–49 percent) clinker phase. The greater injury was thus related to the larger amounts of clinker phase, which in turn resulted in higher and prolonged alkalinity. But Czaja also pointed out that the composition of dusts within the three groups was not consistent, and that, although not yet demonstrated, the constituents of a given dust undoubtedly influence one another.

In short-term experiments of two to three days, Darley [3] dusted the primary leaves of bean plants with fractionated precipitator dust obtained from Germany. The dust contained relatively high amounts of potassium chloride, KCl. When a fine mist was applied to dusted leaves, a portion of the leaf tissue was killed (up to 29 percent) and it was presumed that the action was due to KCl. In later experiments [4] other fractions of the same dust containing very little KCl caused an almost equivalent amount of injury, thus indicating that KCl was apparently not the only factor involved. Current laboratory investigations with different particle-size fractions of precipitator dusts collected around the United States have demonstrated varying degrees of injury to bean leaves when dew is formed on the leaves. There appears to be no effect from dry dusts alone. Inasmuch as these dusts contain very little clinker phase, it is apparent that some components other than those connected directly with hydration of calcium silicates may also be responsible for injury.

(4) *Indirect Effects.*—Pajenkamp [11] reported on unpublished work by Scheffer in Germany during two growing seasons, indicating that even considerable quantities of precipitator dust applied to the soil surface brought about no harmful effects and no other lasting effects on growth or crop yield of oats, rye grass, red clover, and turnips. The dust had a content of about 29.3 percent limestone (analyzed as lime, CaO) and 3.1 percent potassium oxide, K_2O. The maximum rate of deposit was 0.15 mg/cm^2-day. Discontinuous dustings were made at 0.25 mg/cm^2-day to give an average of 0.075 mg/cm^2-day. In one year, the yield of red clover and the weight of turnips were higher in the dusted plots, although the yield of leaves in the latter crop was reduced. Acid manuring of the soil appeared to increase yield but the interaction of dusting and manuring was not understood.

While Scheffer *et al.*[17] found no direct injury to plants, they indicated that there might be indirect effects through changes in soil reaction, which in time might impair yield.

Stratmann and van Haut [18] dusted plants with quantities of dust ranging from 0.1 mg/cm^2-day to 4.8 mg/cm^2-day; dust falling on the soil caused a shift in pH to the alkaline side, which was unfavorable to oats but favorable to pasture grass.

2. Fluorides

Particles containing flouride appear to be much less injurious than gaseous flourides to vegetation. Pack *et al.*[19] reported that 15 percent of gladiolus leaf was killed when plants were exposed four weeks to 0.79 μg/m^3 fluoride as HF, but no necrosis developed when plants were exposed to fluoride aerosol averaging 1.9 μg/m^3 fluoride. Inasmuch as the material was collected from a gas stream which was treated with limestone and hydrated lime, the aerosol was probably calcium fluoride. Moreover, when the accumulated levels of fluoride in leaf tissues were about the same, whether from gas or particulate, injury from the latter was much less.

McCune *et al.*[20] reported an increase of only 4 mm tipburn on gladiolus exposed to cryolite (sodium aluminum fluoride dust), wherein the washed leaf tissue from this treatment showed an accumulation of 29 ppm fluoride. A 70-mm increase in tipburn would have been expected if a similar accumulation had occurred from exposure to HF. Except for the slight tipburn noted above, these authors found that cryolite produced no visible effects on a variety of plants nor did it reduce growth or yield.

It is evident from the work of McCune *et al.*[20] that fluoride in plant tissue is accumulated from cryolite treatment, but the rate of accumulation is much slower than would be expected from a comparable treatment with HF. For example, when comparing washed leaf samples, exposure of gladiolus to HF for three days at $1.01\mu g/m^3$ fluoride resulted in an accumulation of 26.4 ppm fluoride, whereas only 34 ppm was accumulated from an exposure to cryolite for 40 days at $1.7\mu g/m^3$ fluoride. Pack *et al.*[19] reported only one-third as much fluoride accumulated from particulate matter as from gaseous forms.

Both the investigations cited above indicate that much of the particulate matter remains on the surface of the leaf and can be washed off, although that which remains after washing is not necessarily internal fluoride. Reduced phytotoxicity of particulate fluoride is ascribed in part to the inability of the material to penetrate the leaf tissue. In addition, McCune *et al.*[20] suggest that inactivity of particles may be due to their inability to penetrate the leaf in a physiologically active form.

3. Soot

Jennings[21] noted the suggestion that soot may clog stomata and prevent normal gas exchange but that most investigations tend to discount this effect. Microscopic examination failed to show enough clogging of stomata on leaves of shade trees (broad-leaved species) to be significant. He further states that interference with light can be more serious but he offers no data from critical experiments to substantiate this theory.

A well-illustrated report by Berge[22] showed plugged stomata on conifers growing near Cologne, Germany. He also stated that growth was adversely affected.

Necrotic spotting was observed on leaves of several plants where soot from a nearby smokestack had entered a greenhouse.[23] The necrosis was attributed to acidity of the soot particles. Plants outside the greenhouse were not damaged, possibly because the particles had been removed by rain before severe injury could occur.

4. Magnesium Oxide

The possible indirect effect on vegetation of magnesium oxide falling on agricultural soils was reported by Sievers.[24] He noted poor growth in the vicinity of a magnesite-processing plant in Washington. Experiments were designed to grow plants in soil collected at various distances from the processing plant, in normal soil and in soil to which magnesium oxide was added. Suppression of plant growth was demonstrated with the high levels of magnesium. After the processing plant ceased operation, injury to crops in the area became less pronounced, indicating that the injurious effect was not a permanent one.

5. Iron Oxide

Berge,[25] in Germany, dusted experimental plots with iron oxide at the rate of 0.15 mg/cm^2-day over one- to ten-day intervals through the growing season for six years. The plots were planted with cereal grains or turnips, and effects of treatment on the primary product, on straw, and on leaves were noted. No harmful effect of the dust was detected on either crop. There was a tendency for improvement of yields of grain and turnip roots, but this was not statistically significant.

6. Foundry Dusts

Changes in starch reserves were compared in common holly leaves, untreated, and treated with dusts emitted from foundry operations.[6] The critical factor was the amount of light absorbed by the dust layer, and the influence on transpiration or overheating of leaf tissue was of minor significance. These observations agree with some of those reported above on the range of effects of cement-kiln dust on vegetation.

7. Sulfuric Acid Aerosols

These particles too may settle on plants and cause injury. They are not discussed here, however, as the subject is covered in some detail in *Air Quality Criteria for Sulfur Oxides,* a companion document.

C. EFFECTS OF DUSTS ON ANIMALS BY INGESTION OF VEGETATION

Particles which contain chemical components detrimental to animal health may be assimilated through ingestion of plant materials. The toxic components may be absorbed into the plant tissues or may remain as a surface contaminant on the plants. When evaluating the potential harm to animals from ingested vegetation, both absorbed and deposited particles should be considered.

Fluorosis in animals has been reported from ingestion of vegetation covered with a fluoride-containing particulate matter.[26] Injury occurs when absorbed plus deposited fluoride on the plants reached 40 ppm to 50 ppm. Arsenic poisoning of cattle and sheep has occurred from ingestion of arsenic-containing particles settled on vegetation.[27]

D. SUMMARY

There has been relatively little research on the effects of particulate matter on vegetation, and most of the experiments done to date have dealt with specific kinds of dusts rather than the conglomerate mixture normally encountered in the atmosphere.

The significance of dusts as phytotoxicants is not yet entirely clear but there is considerable evidence that certain fractions of cement-kiln dusts adversely affect plants when naturally deposited on moist leaf surfaces. Dry cement-kiln dusts appear to have little deleterious effect, but in the presence of moisture the dust solidifies into a hard adherent crust which can damage plant tissue and inhibit growth. Moderate damage has been observed on the leaves of bean plants dusted at the rate of about 0.47 mg/cm²-day (400 tons/mi²-month) for two days and followed by exposure to naturally occurring dew. Similarly, a marked reduction in the growth of poplar trees one mile from a cement plant was observed after cement production was more than doubled. At levels in excess of 0.1 mg/cm²-day (85 tons/mi²-month), incrustations have been observed on the branches of fir trees, with the result that needles fell prematurely, shortening each succeeding year's flush of growth. Although the mechanism by which injury occurs is not entirely understood, it is possible that the crust intercepts the light needed for photosynthesis and starch formation, causes alkaline damage to tissues, and prevents normal gas exchange in leaf tissues. Injury due to the direct effect of high pH on cell constituents does occur. Plugging of stomata and reduced growth of trees may occur within a short distance of cement plants.

It should be noted, however, that the harmful effect of cement dusts on vegetation is not fully substantiated and has been questioned by some workers. The controversy that surrounds this subject is not surprising in view of the limited research to date. In addition, not all studies have been carried out under identical conditions or with dusts of the same composition. Studies of the effects of cement-kiln dusts deposited on the soil also raise questions. Some investigators report no harmful effects at levels from 0.15 mg/cm²-day to 0.75 mg/cm²-day (130 tons/mi²-month to 640 tons/mi²-month), while others report that concentrations from 0.1 mg/cm²-day to 4.8 mg/cm²-day (86 tons/mi²-month to 4,000 tons/mi²-month) cause shifts in the soil alkalinity which may be favorable to one crop but harmful to another.

Fluorides in particulate form are less damaging to vegetation than gaseous fluorides. Fluoride may be absorbed from depositions of soluble fluoride on leaf surfaces. However, the amount absorbed is small in relation to that entering the plant in gaseous form. The fluoride from particulates apparently has great difficulty penetrating the leaf tissue in a physiologically active form. The research evidence suggests that few if any effects occur on vegetation at fluoride particulate concentrations below about $2\mu g/m^3$. Concentrations of this magnitude can be found in the immediate vicinity of sources of fluoride particulate pollution, but they are rarely found in urban atmospheres. Fluorides absorbed or deposited on plants may be detrimental to animal health. Fluorosis in animals has been reported due to the ingestion of vegetation covered with particulates containing fluorides. In a similar manner arsenic poisoning of cattle and sheep has occurred from ingestion of arsenic-containing particulate that had settled on vegetation.

Soot may clot stomata and may produce necrotic spotting if it carries with it a soluble toxicant, such as one with excess acidity. Magnesium oxide deposits on soils have been shown to reduce plant growth, while iron oxide deposits appear to have no harmful effects and may be beneficial. Sulfuric acid aerosols will cause leaf spotting. The levels at which these materials may produce a toxic response are not well defined.

E. REFERENCES

1. Peirce, G. J. "An Effect of Cement Dust on Orange Trees." Plant World, Vol. 13, pp. 283–288, 1910.
2. Parish, S. B. "The Effects of Cement Dust on Citrus Trees." Plant World, Vol. 13, pp. 288–291, 1910.
3. Darley, E. F. "Studies on the Effect of Cement-Kiln Dust on Vegetation." J. Air Pollution Control Assoc., Vol. 16, pp. 145–150, 1966.
4. Darley, E. F. Unpublished data.
5. Czaja, A. T. "Uber das Problem der Zementstaubwirkungen auf Pflanzen." Staub, Vol. 22, pp. 228–232, 1962.
6. Bohne, H. "Schadlichkeit von Staub aus Zimentwerken für Waldbestande." Allgem. Forstz., Vol. 18, pp. 107–111, 1963.
7. Steinhubel, G. "Zmeny v skrobovych rezervach listov cezminy po umelom znecisteni pevnym popraskom." Biologia, Vol. 18, pp. 23–33, 1962. (Abstract, in German, pp. 32–33, with the title: Veranderungen in den Starkereserven der Blatter der gemeinen Stechpalme nach einer künstlichen Verunreinigung durch Staub.)
8. Czaja, A. T. "Uber die Einwirkung von Stauben, speziell von Zementofenstaub auf Pflanzen." Angew. Botan., Vol. 40, pp. 106–120, 1966.
9. Anderson, P. J. "The Effect of Dust from Cement Mills on the Setting of Fruit." Plant World, Vol. 17, pp. 57–68, 1914.
10. Schonbeck, H. "Beobachtungen zur Frage des Einflusses von industriellen Immissionen auf die Krankbereitschaft der Pflanze." Berichte der Landesanstalt für Bodennutzungsschutz (Bochum), Vol. 1, pp. 89–98, 1960.
11. Pajenkamp, H. "Einwirkung des Zementofenstaubes auf Pflanze and Tiere." Zement-Kalks-Gips, Vol. 14, pp. 88–95, 1961.
12. Raymond, V. and Nussbaum, R. "A propos des poussieres de cimenteries et leurs effets sur l'homme, les plants, et les animaux." Pollut. Atmos. (Paris), Vol. 3, pp. 284–294, 1966.
13. Guderian, R. and Pajenkamp, H. "Einwirkung des Zementofenstaubes auf Pflanzen und Tiere." Staub, Vol. 21, pp. 518–519, 1961.
14. Wentzel, K. F. and Pajenkamp, H. "Einwirkung des Zementofenstaubes auf Pflanzen und Tiere." Zeit. für Pflanzenkrankh, Vol. 69, p. 478, 1962.
15. Czaja, A. T. "Die Wirkung von verstaubtem Kalk und Zement auf Pflanzen." Qualitas Plant Mater. Vegetabiles, Vol. 8, pp. 154–212, 1961.
16. Czaja, A. T. "Zementstaubwirkungen auf Pflanzen: Die Entstehung der Zementkrusten." Qualitas Plant Mater. Vegetabiles, Vol. 8, pp. 201–238, 1961.
17. Scheffer, F., Przemeck, E., and Wilms, W. "Untersuchungen über den Einfluss von Zementofen-Flugstaub auf Boden and Pflanze." Staub, Vol. 21, pp. 251–254, 1961.
18. Stratmann, H. and van Haut, H. "Vegetationsversuche mit Zementflugstaub." (Unpublished investigations of the Kohlen-Stoff-biologischen Forschungsstation) Essen, Germany, 1956.
19. Pack, M. R., Hull, A. C., Thomas, M. D., and Transtrum, L. G. "Determination of Gaseous and Particulate Inorganic Fluorides in the Atmosphere." Am. Soc. Testing Mater., Spec. Tech. Pub. 281, 1959, pp. 27–44.
20. McCune, D. C., Hitchcock, A. E., Jacobson, J. S., and Weinstein, L. H. "Fluoride Accumulation and Growth of Plants Exposed to Particulate Cryolite in the Atmosphere." Contrib. Boyce Thompson Inst., Vol. 23, pp. 1–22, 1965.
21. Jennings, O. E. "Smoke Injury to Shade Trees." Proc. Nat. Shade Tree Conf., 10th, 1934, pp. 44–48.
22. Berge, H. "Luftverunreinigungen im Raume Köln." Allgem. Forstz., Vol. 51–52, pp. 834–838, 1965.
23. Miller, P. M. and Rich, S. "Soot Damage to Greenhouse Plants." Plant Disease Reptr., Vol. 51, p. 712, 1967.
24. Sievers, F. J. "Crop Injury Resulting from Magnesium Oxide Dust." Phytopathology, Vol. 14, pp. 108–113, 1924.
25. Berge, H. "Emissionsbedingte Eisenstaube und ihre Auswirkungen auf Wachstum und Ertrag landwirtschaftlicher Kulturen." Zeit. Luftvereinigung (Düsseldorf), Vol. 2, pp. 1–7, 1966.
26. Shupe, J. L., Miner, M. L., Harrison, L. E., and Greenwood, D. A. "Relative Effects of Feeding Hay Atmospherically Contaminated by Fluoride Residues, Normal Hay Plus Calcium Fluoride, and Normal Hay Plus Sodium Fluoride to Dairy Heifers." Am. J. Vet. Res., Vol. 23, pp. 777–787, 1962.
27. Phillips, P. H. "The Effects of Air Pollutants on Farm Animals." In: Air Pollution Handbook, P. L. Magill, F. R. Holden, and C. Ackley (eds.), McGraw-Hill, New York, 1956.

Chapter 7

SOCIAL AWARENESS OF PARTICULATE POLLUTION

Table of Contents

Page

A. INTRODUCTION 99

B. THE STUDIES 99
  1. St. Louis, Missouri 99
  2. Nashville, Tennessee 100
  3. Birmingham, Alabama 101
  4. Buffalo, New York 101

C. SUMMARY 102

D. REFERENCES 102

List of Figures

Figure

7–1 Proportion of Population in St. Louis Stating Air Pollution Present in Their Area of Residence, and Proportion of St. Louis Population Bothered 100

7–2 The Level of Air Pollution Related to Public Opinion in Three Income Groups (Nashville, Tennessee) 101

7–3 Radial Distribution from the Center of Nashville of Air Pollution Levels, Socioeconomic Status and Public Concern About Air Pollution 101

Chapter 7

SOCIAL AWARENESS OF PARTICULATE POLLUTION

A. INTRODUCTION

The nuisance of air pollution is to some extent a subjective perception, and its significance is therefore influenced to some extent by the way the public feels about pollution. Nevertheless, it is acceptable practice to conclude that nuisance and levels of pollution are related if it can be demonstrated that a nuisance response by a sample population is positively related to average levels of actual pollution. The development of such evidence is difficult because public reaction probably reflects pollution peaks rather than the mean value for an extended time period. This problem has been considered by McKee[1] in a recent paper.

Several studies have attempted to assess the annoyance to a population of community air pollution, but not all have taken advantage of available aerometric data to establish the relationships between air pollution levels and attitudes and opinion among the affected population. The chief value of these surveys lies in demonstrating that a significant proportion of the public is aware of, and concerned about, air pollution, and is willing to act to abate the nuisance.[2, 3] One of the most obvious indications of the nuisance of air pollution is citizen complaints. Generally speaking, the person who is willing to take the time to telephone or write a complaint about air pollution is probably seriously irritated by it. And those who actually complain may represent only the top of the iceberg. There are many who may be irritated who will not complain because they do not believe complaining will do any good. There are others, conspicuous by their absence, who have moved away because of the effects of air pollution on their health, or because of the aesthetic degradations of their neighborhood. Unfortunately, very little work has been done in this particular area of measuring the response of the public to the nuisance of air pollution.

It is important to note that application of present control technology may result in a much larger reduction in larger particles than in smaller particles. The remaining smaller particles will not be as readily discernible to the public, and awareness of particulate pollution may diminish.

The effect of particulates on the total ecological system and man's enjoyment of his environment cannot as yet be fully evaluated.

B. THE STUDIES

1. St. Louis, Missouri

One major investigation of the relationship between public opinion and particulate air pollution concentrations is that conducted in the Greater St. Louis area.[3, 4] This area comprises portions of St. Louis County, Missouri, and portions of Madison and St. Clair Counties, Illinois. It includes St. Louis, East St. Louis, and Granite City, and thus most of the population residing in the St. Louis metropolitan area, as defined by the Bureau of the Census. Persons interviewed were asked whether they believed that air pollution was present and whether they regarded it as a nuisance. The responses were related to the pollution level measured both as suspended particle concentration and as soiling index. (See Chapter 1 for a discussion of this latter measure of pollutant concentration.) Figure 7–1 shows the results obtained for both questions in terms of suspended particle concentrations. It will be seen that the population becomes aware of pollution before it regards it as a nuisance. Where the average annual geometric mean of particles was 80 $\mu g/m^3$, better than 30 percent of the popula-

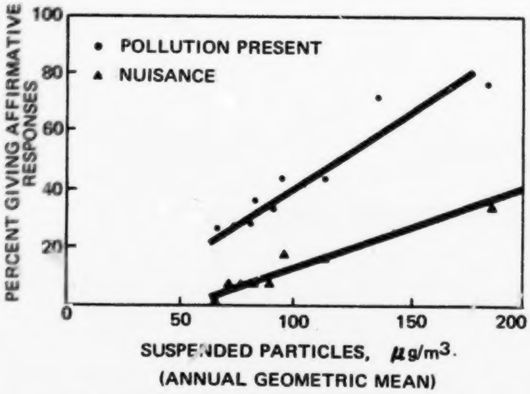

FIGURE 7-1. Proportion of Population in St. Louis Stating Air Pollution Present in Their Area of Residence, and Proportion of St. Louis Population Bothered. (This figure presents the results obtained from interviews.)

tion indicated awareness of air pollution. Fifty percent and 75 percent of the survey population were aware of air pollution in the area where the average annual levels of particulate matter were 120 $\mu g/m^3$ and 160 $\mu g/m^3$ respectively. None of the population appeared to be bothered by particle concentrations below 50 $\mu g/m^3$, 10 percent of the population was bothered at a level of about 80 $\mu g/m^3$, 20 percent was bothered about 120 $\mu g/m^3$, 33 percent at 160 $\mu g/m^3$, while 40 percent regarded a concentration of 200 $\mu g/m^3$ as constituting a nuisance. The data on the nuisance response in Figure 7-1 can be expressed approximately in the form

$$y \simeq 0.3x - 14$$

over the range studied, where y is the percentage of the population expressing dissatisfaction, and x is the annual geometric mean of suspended particle concentration in $\mu g/m^3$. The equation assumes that the character of the pollution is the same for areas of both high and low pollution.

2. Nashville, Tennessee

A study by Smith *et al.*[5] reveals the complexity of public opinion surveys. The method used was similar to that in other surveys: public opinion data were compared with aerometric data from the nearest air sampling station. One group of questions in the public opinion survey dealth specifically with nuisance aspects of air pollution, asking respondents to indicate whether the outside of the house got too dirty, whether automobiles got dirty too fast, and whether too much dust collected on porches and window sills. Another question, measuring "bother" by air pollution was embedded in a series of questions relating to health, and responses to it should not be compared directly with responses of general concern and nuisance in other surveys. The proportion of affirmative responses to the question concerning too much dust and dirt on porch and window sills showed a clear increase with increasing particle concentrations. The data do not, however, permit a predictable quantitative statement of this responsibility.

Survey results indicated that up to 3.8 percent of the respondents voluntarily expressed awareness and concern about air pollution as a health problem; an extension of the sample population of about 2,850 to the total population indicated that this figure equated to approximately 9,000 residents. In response to a direct question, 23 percent of the respondents—or approximately 50,000 residents, if the sample were projected to the total population—stated they were bothered in some way by air pollution. In response to direct questions, several specific non-health aspects of smog—soiling, decreased visibility, odors, and property damage—were cited as affecting from 18 percent to 51 percent of the respondents, or between 40,000 to 100,000 of all residents, as based on the sample population. It was also concluded that the frequency of days of acute pollution had an additional influence on the proportion of people who would express concern and annoyance.

The study indicated some relationships between level of concern and socioeconomic status. At high levels of air pollution, concern was greater among women of high socioeconomic status than among women of low socioeconomic status, although at low levels of air pollution, those of low socioeconomic status expressed more concern than those of high status. There was also a relationship between socioeconomic status and the distance of residence from the center of Nashville (the more affluent living farther away in general). Figures 7-2 and 7-3 together demonstrate the interlocking relationship; Figure 7-2 uses arbitrary scales for both variables and should not be taken to indicate the exact form of the relationships.

3. Birmingham, Alabama

Stalker and Robison [6] compared data from 21 air sampling stations with public responses from a sample of 7200 households. Data were gathered only from people living within one mile of an air sampling station. The investigators found that 10 percent of the population believed a nuisance existed at a seasonal (summer) mean suspended particle concentration between 60 μg/m³ and 180 μg/m³, and more than 30 percent of the population believed a nuisance existed at levels between 100 μg/m³ and 220 μg/m³. The authors use their dustfall data to demonstrate that for each increment of 10 tons/mile²-month another 10 percent of the affected public would become concerned and consider that level a general nuisance. Although the published data indicates that the relationship probably is not linear, they do indicate that the respondents clearly defined a nuisance situation in their area of residence at annual mean dustfall values of 10 tons/mile²-month or above.

4. Buffalo, New York

de Groot and Samuels [2] used two independent samples taken three years apart to determine public opinion. Opinions were assessed against aerometric data from the nearest of nine air sampling stations. The measure of public opinion was the percentage of the population considering air pollution a serious community problem. The investigators counted only clear, unequivocal responses; respondents who indicated uncertainty about the seriousness of the problem were assigned to the "not serious" category. In Buffalo, as in Birmingham, there were apparent positive relationships between public awareness and levels both of dustfall and of

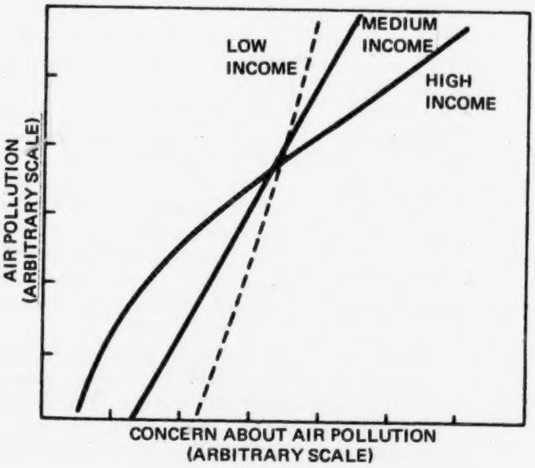

Figure 7-2. The Level of Air Pollution Related to Public Opinion in Three Income Groups (Nashville, Tennessee).[5] (Figures 7-2 and 7-3 show the interlocking relationships between several parameters affecting public concern over air pollution in Nashville. At high levels of pollution, concern is strongest in those of highest socio-economic status, while at low pollution levels, concern is greatest among those of low status. This result is possibly connected with the radial distance from Nashville at which persons of different socioeconomic status reside.)

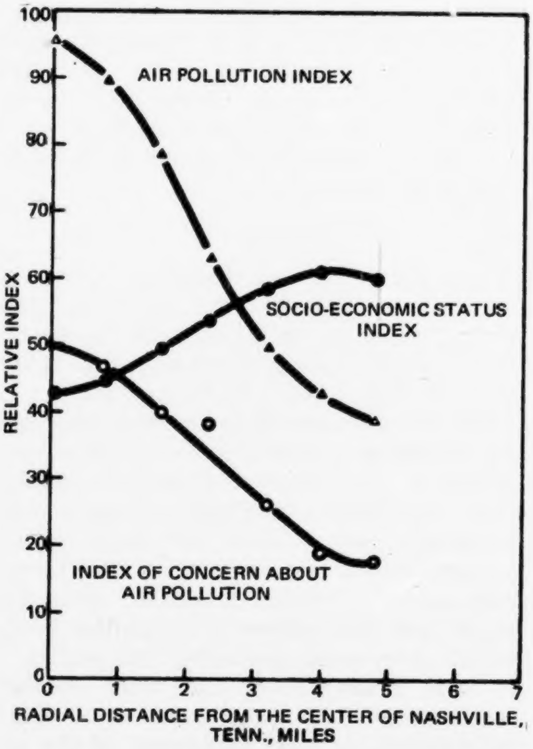

Figure 7-3. Radial Distribution from the Center of Nashville of Air Pollution Levels, Socioeconomic Status and Public Concern About Air Pollution.[5]

suspended particles. There was also a positive relationship between the frequency of days of acute levels of these pollutants and public opinion.

C. SUMMARY

Public opinion survey data in several cities indicate a positive relationship between the concern expressed about air pollution by a population and the actual levels of particulate pollution—used as an index of air pollution. In general, over the ranges studied, an increasing proportion of the population expresses dissatisfaction over air pollution as concentrations of particulate matter increase. However, while the several studies agree, for the most part, in this qualitative relationship, it is difficult to compare the studies quantitatively. Each study used different sampling schemes, questionnaire schedules, and methods of interviewing. In addition, the socioeconomic characteristics of the population sampled varied from city to city. The St. Louis data has been singled out for quantitative expression because it showed the most consistent association between air pollution and public awareness. However, it is intended to serve only as an example.

Over the approximate range 50 $\mu g/m^3$ to 200 $\mu g/m^3$, the expression

$$y \simeq 0.3x - 14$$

relates roughly the percentage of concerned St. Louis population, y, and the annual geometric mean suspended particle concentration x ($\mu g/m^3$). Thus, 10 percent of the study population was bothered by air pollution in areas where the annual geometric mean value of suspended particulates was about 80. $\mu g/m^3$. About 20 percent of the study population was bothered in areas with an annual geometric mean value of 120 $\mu g/m$ and 33 percent with a mean of 160 $\mu g/m^3$. The responses are probably associated with the short term fluctuations in particulate levels which underlie any averaging time period.

Other studies show that when dustfall levels exceeded an annual mean of 10 tons/mile^2-month, at least 10 percent of the affected population expressed concern about a nuisance situation.

The available literature also indicates that the extent to which a population considers air pollution an annoyance is related to the frequency of days with acute pollution as well as to the average level that the level of concern is related to socioeconomic status, and that the population becomes aware of pollution at particle concentrations lower than those at which they consider that it constitutes a nuisance.

In the St. Louis study, 30 percent of the study population were aware of pollution in areas where the annual geometric mean value of suspended particulates was 80 $\mu g/m^3$, 50 percent in areas with 120 $\mu g/m^3$ and 75 percent in areas with 160 $\mu g/m^3$.

D. REFERENCES

1. McKee, H. C. "Why a General Standard for Particulates?" (Presented at the Symposium on Air Quality Standards: The Technical Significance, 156th National Meeting, American Chemical Society, September 12, 1968.)
2. de Groot, I. and Samuels, S. W. "People and Air Pollution: A Study of Attitude in Buffalo, New York. An Interdepartment Report." New York State Dept. of Health, Air Pollution Control Board, 1965. (See also de Groot, I., Loring, W., Rihm, A., Samuels, S. and Winkelstein, W., same title, J. Air Pollution Control Assoc., Vol. 16, pp. 245–247, 1966.)
3. "Public Awareness and Concern with Air Pollution in the St. Louis Metropolitan Area." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Washington, D. C., May 1965. (See also Schusky, J., same title, J. Air Pollution Control Assoc., Vol. 16, pp. 72–76, 1966.)
4. Williams, J. D. and Bunyard, F. L. "Interstate Air Pollution Study, Phase II Project Report, Vol. VII—Opinion Surveys and Air Quality Statistical Relationships." U.S. Dept. of Health, Education, and Welfare, Div. of Air Pollution, Cincinnati, Ohio, 1966.
5. Smith, W. S., Schueneman, J. J., and Zeidberg, L. D. "Public Reaction to Air Pollution in Nashville, Tennessee." J. Air Pollution Control Assoc., Vol. 14, pp. 418–423, 1964.
6. Stalker, W. W. and Robison, C. B. "A Method for Using Air Pollution Measurements and and Public Opinion to Establish Ambient Air Quality Standards." J. Air Pollution Control Assoc., Vol. 17, pp. 142–144. 1967.

Chapter 8

ODORS ASSOCIATED WITH ATMOSPHERIC PARTICULATE MATTER

Table of Contents

Page

A. INTRODUCTION 105

B. EVIDENCE FOR THE ASSOCIATION OF ODOR WITH PARTICLES 105

C. HYPOTHETICAL MECHANISM FOR THE ASSOCIATION OF ODOR WITH PARTICLES 106
  1. Volatile Particles 106
  2. Desorption of Odorous Matter by Particles 106
  3. Odorous Particles 107

D. COMMON ODOR PROBLEMS 107

E. SUMMARY 107

F. REFERENCES 108

List of Tables

Table

8–1 Most Frequently Reported Odors 107

Chapter 8

ODORS ASSOCIATED WITH ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

The human olfactory sense has the ability to detect and respond to thousands of different materials or chemical compounds. Some odorants can be detected in concentrations as low as one part per billion. Modern instrumentation lacks both the selectivity and the sensitivity possessed by the olfactory sense for the detection of many odorants.

Odorants in themselves may not cause organic disease; however, the discomfort and disagreeableness that may be brought about by obnoxious odors can cause some temporary ill effects. The effects that border upon ill health include lowered appetite, lowered water consumption, impaired respiration, nausea, vomiting, and insomnia.

Particulate matter in itself is not considered to be capable of directly stimulating the olfactory sense. However, this should not be interpreted to mean that all airborne particulate matter, as a pollutant category, is incapable of stimulating the odor sense nor that particles cannot be involved in the delivery of the odorant to the receptor cells. There is evidence that some particles can stimulate the sense of smell because the particle itself is volatile or because this particle is desorbing a volatile odorant.[1] There is also speculation[2] that some particulate matter is capable of stimulating the sense of smell. Regardless of the mechanism of how particles are involved in the stimulation of the olfactory sense, the important fact is that they definitely appear to be involved.

B. EVIDENCE FOR THE ASSOCIATION OF ODOR WITH PARTICLES

The idea that odors are associated with particles is supported by observations that filtration of particles from an odorous air stream can reduce the odor level. Rossano and Ott[2] showed that the removal of particulate matter from diesel exhaust by thermal precipitation effected a marked reduction in odor intensity. The precipitation method was selected because it provides minimal contact between the collected particles and the gaseous components of the diesel exhaust stream. Thus, effects that could be produced by a filter bed, such as removal of odorous gases by absorption in the filter cake, are eliminated. The observed odor reduction must, therefore, have resulted directly from the removal of particulate matter. The particles collected by Rossano and Ott were aggregates of spherical balls about 0.04μ to 0.05μ in diameter. Particulate matter from diesel exhaust collected on glass fiber filters by Linnell and Scott[3] yielded a heavy "diesel" odor. Analysis of the particulate matter showed that it contained no acrolein or formaldehyde, although it did release NO_2 on being heated to 100°C. The authors conclude that "no appreciable gas phase concentration changes for acrolein or formaldehyde will result from particulate removal." Other more or less casual observations on the role of particulate matter in community odor nuisance problems appear occasionally in the literature.[4]

A second piece of evidence that implicates the association of particles with odors is the production of a mild odor, sometimes described as "yeasty," in air that is passed through a bed of activated carbon. Turk and Bownes[5] have shown that this odor is not produced by any detectable desorption of gaseous matter from the carbon, and that the same odor can be produced by passing air through silica gel.[6] It is possible that subfilterable particles are associated with this phenomenon.

Evidence for the association of particulate matter with outdoor odors has sometimes been sought in the gross errors in odor intensities preducted on the basis of gas-phase dispersion of an odor source. However, the interpretation of these experiments is open to doubt, and it is probable that the effects are a result of fortuitous fluctuations in emission. For example, Walter and Amberg [7] have found that concentrations of hydrogen sulfide in a kraft paper pulp mill recovery furnace can vary from 100 ppm to as much as 1,000 ppm. Such wide variations in emission may help to explain the discrepancy, reported by Wohlers [8] for a kraft mill, between the odor intensities observed and those predicted from odor threshold concentrations and dilutions calculated by Sutton's formula.[9] The likelihood that errors in calculations of the atmospheric dispersal of true gases are comparatively small is supported by experiments with gas tracers like sulfur hexafluoride (SF_6). Collins *et al.*[10] and Turk *et al.*[11] have shown agreement between calculated and observed concentrations of a tracer gas at distances up to about three miles from the emission point to be within about 25 percent. It is true that olfactory sensation is responsive to concentrations that may be present for only brief intervals, whereas dispersion calculations refer to time averages for intervals of about one-half hour, and the tracer gas studies cited used 20-minute integrated samples. Some of the discrepancy between results of odor measurements on the one hand, and results from calculations or tracer gas tests on the other, may therefore be accounted for by the peak-to-mean ratios that result from the effects of turbulence and eddies in the air stream. There are no published data on the minimum time duration required for odor detection or odor nuisance responses. Efforts by Turk and coworkers to determine the ratio of "instantaneous" (one second) to average (20 minute) concentrations of tracer gas failed to show significantly high values, possibly because such occurrences are rare and escaped the sampling grid. In any event, the peak-to-mean ratios would have to be in the 1,000 to 10,000-fold range to explain observed discrepancies, and we have no data that are based on meteorological factors alone to support such extreme values.

C. HYPOTHETICAL MECHANISMS FOR THE ASSOCIATION OF ODOR WITH PARTICLES

1. Volatile Particles

Liquid or even solid aerosols may be sufficiently volatile that their vaporization on entering the nasal cavity produces enough gaseous material to be detected by smell. Such aerosols may be relatively pure substances, such as particles of camphor, or they may be mixtures which release volatile components. The retention of the odorous properties of volatile aerosols will, of course, depend on the prevailing temperature and on the length of time they are dispersed in air. In a cold atmosphere, the relatively greater temperature rise accompanying inhalation will accelerate the production of gaseous odorant.

2. Desorption of Odorous Matter by Particles

Goetz [12] has treated the kinetics of the interaction between gas molecules and the surface of airborne particles. His theoretical considerations were directed to the question of transfer of toxicants by particles, but are also applicable to odors. Even if a given aerosol is intrinsically odorless, it could act as an odor intensifier if: 1. the sorptive capacity of the aerosol particles for the odorant were smaller than the affinity of the odorant for the nasal receptor and at the same time, 2. the sorptive capacity of the aerosol particles were large enough to produce an accumulation of odorant on the particle surface. Such aerosol particles would concentrate odorous molecules on their surfaces, but the odorous matter would be transferred to olfactory receptors when the aerosol entered the nasal cavity. The odorous matter would then be present at the receptor sites in concentrations higher than in the absence of the aerosol. The resulting effect would be synergistic. See Chapter 10–C–3 for a discussion of synergistic effects of particles and irritants. These synergistic effects may be analogous to particle-odorant synergism. If an odorant is more strongly adsorbed by the aerosol particles than by the olfactory receptors, transfer of the odorant to the receptors would be impeded and the particles would actually attenuate the odor.

3. Odorous Particles

No study has ever rigorously defined the upper limit of particle size for airborne odorous matter. Particles up to about 8 or 10×10^{-4} μ in diameter are considered to be molecules that can exist in equilibrium with a solid or liquid phase from which they escape by vaporization. The vapor pressure decreases as the molecular weight increases, and particles above about 10^{-3} μ do not generally exist in any significant concentration in equilibrium with a bulk phase; hence we do not consider them to be "vapors." Nonetheless, it is possible that odorant properties do not disappear when particle sizes exceed those of vapor molecules. Our knowledge about particles in the size range of 1 to 5×10^{-2} μ (up to about the size of small viruses) is relatively meager, and we do not know whether or not they can be odorous, nor what the effect of an electrical charge on their odorous properties might be. Larger particles may also be intrinsically odorous, although their more significant role may be to contribute to odor by absorbing and desorbing odorous gases and vapors.

D. COMMON ODOR PROBLEMS

Kerka and Kaiser[13] surveyed State and local air pollution control personnel and compiled the list of most frequently reported odors shown in Table 8-1.

Of the 35 listings in Table 8-1, nine are either known to be or suspected to be particulate-borne. These nine include gasoline- and diesel-engine exhaust, coffee roasting, restaurant odors, paint spraying, roofing and street paving, asphalt manufacturing, home incinerators and backyard trash fires, city incinerator burning garbage, and open-dump fires.

E. SUMMARY

Airborne particulate matter, as an air pollutant category, is normally not considered as a source of odor stimulation. However, there is evidence that some particulates having volatile components can produce an odor response in human receptors.

Further, by a suggested mechanism of adsorption and subsequent desorption, an odorant may be transferred by a particulate sub-

Table 8-1.—MOST FREQUENTLY REPORTED ODORS

Source of odor	*Number reported*
Animal odors:	
Meat packing and rendering plants	12
Fish oil odors from manufacturing plants	5
Poultry ranches and processing	4
Odors from combustion processes:	
Gasoline and diesel engine exhaust	10
Coke-oven and coal-gas odors (steel mills)	8
Maladjusted heating systems	3
Odors from food processes:	
Coffee roasting	8
Restaurant odors	4
Bakeries	3
Paint and related industries:	
Manufacturing of paint, lacquer, and varnish	8
Paint spraying	4
Commercial solvents	3
General chemical odors	
Hydrogen Sulfide	7
Sulfur Dioxide	4
Ammonia	3
General industrial odors	
Burning rubber from smelting and debonding	5
Odors from dry-cleaning shops	5
Fertilizer plants	4
Asphalt odors—roofing and street paving	4
Asphalt odors—manufacturing	3
Plastic manufacturing	3
Foundry odors	
Core-oven odors	4
Heat treating, oil quenching, and pickling	3
Smelting	2
Odors from combustible waste:	
Home incinerators and backyard trash fires	4
City incinerators burning garbage	3
Open-dump fires	2
Refinery odors:	
Mercaptans	3
Crude oil and gasoline odors	3
Sulfur	1
Odors from decomposition of waste:	
Putrefaction and oxidation—organic acids[a]	3
Organic nitrogen compounds—decomposition of protein[a]	2
Decomposition of lignite (plant cells)	1
Sewage odors:	
City sewers carrying industrial waste	3
Sewage treatment plants	2

[a] Probably related to meat processing plants.

strate. When one examines the various types of odor sources which result in public awareness of an undesirable situation, a significant number of these listings are either known to be or suspected to be particulate-borne. A survey of State and local air pollution control officials revealed that approximately one-fourth of the most frequently reported odors are those which are known to be, or are suspected to be, associated with particulate air pollution. The sources of these odorous particles are diverse, including diesel- and gasoline-engine exhaust, coffee-roasting operations, paint spraying, street paving, and the burning of trash. Despite the absence of an exact mechanism to explain the association of odor with particulates, their intimate involvement in multiple categories of citizen nuisance complaints cannot be ignored.

F. REFERENCES

1. Roderick, W. R. "Current Ideas on the Chemical Basis of Olfaction." J. Chem. Educ., Vol. 43, pp. 510–520, 1966.
2. Rossano, A. T. and Ott, R. R. "The Relationship Between Odor and Particulate Matter in Diesel Exhaust." Preprint. (Presented at the Annual Meeting of the Pacific Northwest International Section, Air Pollution Control Association, Portland, Oregon, November 5–6, 1964.)
3. Linnel, R. H. and Scott, W. E. "Diesel Exhaust Composition and Odor Studies." J. Air Pollution Control Assoc., Vol. 12, pp. 510–515, 1962.
4. Quebedeaux, W. A. "New Applications for Industrial Odor Control." Air Repair, Vol. 4, pp. 141–142, 170–171, 1954.
5. Turk, A. and Bownes, K. "Inadequate Stimulation of Olfaction." Science, Vol. 114, pp. 234–236, 1951.
6. Turk, A., Kakis, F., and Morrow, J. "The Adsorbent Odor." Preprint. (Presented at the American Chemical Society meeting, New York, Sept. 1960.)
7. Walther, J. E. and Amberg, H. R. "Continuous Monitoring of Kraft Mill Stack Gases with a Process Gas Chromatograph." Tappi, Vol. 50, pp. 19–23, 1967.
8. Wohlers, H. C. "Odor Intensity and Odor Travel from Industrial Sources." Intern. J. Air Water Pollution, Vol. 7, pp. 71–78, 1963.
9. Sutton, O. G. "The Theoretical Distribution of Airborne Pollution from Factory Chimneys." Quart. J. Roy. Meteorol. Soc., Vol. 73, pp. 426–436, 1947.
10. Collins, G. F., Barlett, F. E., Turk, A., Edmonds, S. M. and Mark, H. "A Preliminary Evaluation of Gas Air Tracers." J. Air Pollution Control Assoc., Vol. 15, pp. 109–112, 1965.
11. Turk, A., Edmonds, S. M., Mark H. L., and Collins, G. F. "Sulfur Hexafluoride as a Gas-Air Tracer." Environ. Sci. Technol., Vol. 2, pp. 44–48, 1968.
12. Goetz, A. "On the Nature of the Synergistic Action of Aerosols." Intern. J. Air Water Pollution, Vol. 4, pp. 168–184, 1961.
13. Kerka, W. F. and Kaiser, E. R. "An Evaluation of Environmental Odors." J. Air Pollution Control Assoc., Vol. 7, pp. 297–301, 1958.

Chapter 9

THE RESPIRATORY SYSTEM: DEPOSITION, RETENTION, AND CLEARANCE OF PARTICULATE MATTER

Table of Contents

Page

A. INTRODUCTION 111

B. ANATOMY OF THE HUMAN RESPIRATORY TRACT 111

C. FACTORS AFFECTING THE DEPOSITION AND RETENTION OF PARTICULATE MATTER IN THE RESPIRATORY SYSTEM 112
- 1. Physical-Mathematical Treatments 112
  - a. Deposition Mechanisms 112
  - b. Aerodynamic Factors 113
  - c. The Models 114
- 2. Experimental Studies of Factors Affecting Deposition and Retention 116
  - a. Total Deposition in the Respiratory Tract 117
    - (1) Effect of Particle Size 117
    - (2) Physiological Parameters 117
  - b. Regional Deposition 118
    - (1) Nasal Fractionation 118
    - (2) Lung Deposition 118
    - (3) Comparative Human and Animal Retention 119
- 3. Conclusions Reached about Alveolar Deposition 119

D. FACTORS AFFECTING THE CLEARANCE OF PARTICULATE MATTER FROM THE RESPIRATORY SYSTEM 119
- 1. Clearance Model 120
- 2. Clearance from the Tracheobronchial System 121
- 3. Clearance from the Alveolar Surface 122

E. SUMMARY 123

F. REFERENCES 123

List of Figures

Figure

9–1 The Major Anatomical Features of the Human Respiratory System 112

9–2 The Terminal Bronchial and Alveolar Structure of the Human Lung 112

9–3 Calculated Fraction of Particles Deposited in the Respiratory Tract as a Function of Particle Radius 114

9–4 Fraction of Particles Deposited in the Three Respiratory Tract Compartments as a Function of Particle Diameter 115

9–5 Data from Figure 9–4 for the Nasopharyngeal Compartment Plotted as a Log-probability Function (Minute Volume: 20 l/min) 116

9–6 Data from Figure 9–4 for the Pulmonary Compartment Plotted as a Log-probability Function (Minute Volume: 20 l/min) 116

9–7 Schematic Portrayal of Dust Deposition Sites and Clearance Processes 120

List of Tables

Table

9–1 Respiratory Airflow Patterns for a Group of Healthy Young Men 113

Chapter 9

THE RESPIRATORY SYSTEM: DEPOSITION, RETENTION, AND CLEARANCE OF PARTICULATE MATTER

A. INTRODUCTION

In urban communities, exposure to atmospheric pollutants may constitute a health hazard that is the most serious single consequence of air pollution. Chapters 10 and 11 discuss the effects of particulate pollutants on health in terms of toxicological and epidemiological studies. Pollutants are likely to enter the human body mainly via the respiratory system; other routes of entry are of minor importance. Damage to the respiratory organs may follow directly, or the pollutant may be transported by some mechanism to a remote susceptible organ. It is apparent that a study of the effect on health of particulate atmospheric pollutants requires an understanding of the mechanisms and efficiencies of deposition of particles in the respiratory system, and of the subsequent retention within, and clearance from the system, as well as its secondary relocation to other sites in the body. This chapter provides a brief introduction to the physics and physiology of deposition, retention, and clearance in the respiratory system. More complete descriptions may be found in several reviews.[1–3]

Experimental studies of the several factors involved in deposition, retention, and clearance processes have been backed up by theoretical treatments and, in the discussion which follows, descriptions of these theoretical models precede those of experimental work. One of the latest theoretical models is that developed by the Task Group on Lung Dynamics for Committee II of the International Radiological Protection Commission.[4] The Task Group's report establishes the usefulness of the Stokes (mass median) diameter (cf. Chapter 1–A) of a particle as a measure of deposition probability. This is of practical importance, since the Stokes diameter may readily be determined under field conditions.

The anatomy of the respiratory system plays an important part in determining the effects of inhaled particles, and a very short description of this anatomy is provided. Several extensive presentations of morphological studies are available,[5, 6] and Davies [7] gives a formalized concept of the anatomy of the human respiratory tract.

B. ANATOMY OF THE HUMAN RESPIRATORY TRACT

The respiratory system is usefully broken down into three main sections:

1. the nasopharyngeal structure;
2. the tracheobronchial system; and
3. the pulmonary structure, within which oxygen and carbon dioxide are exchanged between respired air and blood.

Figure 9–1 shows the location of these features. The nasal passages lead, via the nasopharyngeal structure and the larnyx, to the trachea and the bronchi, which are made up of 23 generations of dichotomous branching tubes terminating in the alveolar (air) sacs. Figure 9–2 is a schematic representation in greater detail of the terminal bronchiole and pulmonary structure.

Estimates of numbers of alveoli differ somewhat from one experimenter to another: a recent suggestion [8] is that there are 300 million, together with 14 million alveolar ducts. The alveoli are probably between 150μ and 400μ in diameter, so that the total alveolar surface varies between about 30 m^2 and 80 m^2. The increasing total cross-sectional area with progression down the respiratory tract

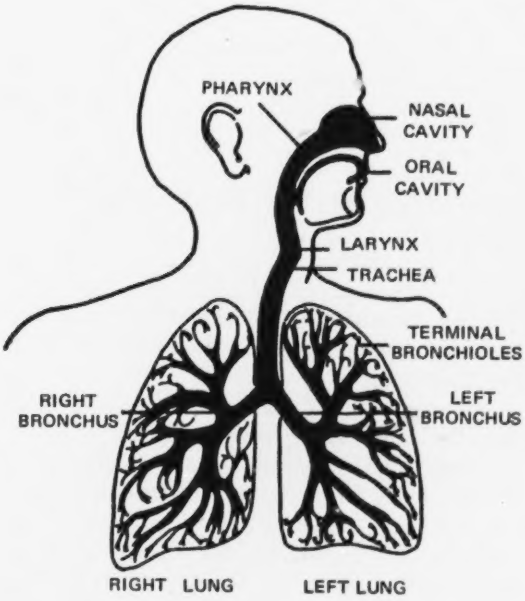

FIGURE 9–1. The Major Anatomical Features of the Human Respiratory System. (The diagram shows the major divisions of the human respiratory tract into nasopharyngeal, tracheobronchial, and pulmonary compartments.)

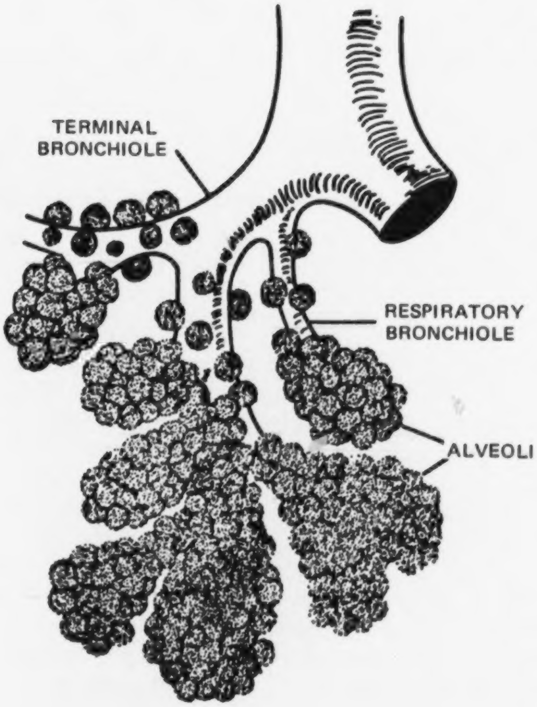

FIGURE 9–2. The Terminal Bronchial and Alveolar Structure of the Human Lung. (The diagram shows the pulmonary structure of the respiratory tract.)

leads to a marked decrease in the velocity of air movement with depth.

The nasopharyngeal and tracheobronchial structures possess ciliated epithelium covered with mucus arising from goblet cells and secretory glands. These structures also make up the anatomical "dead space," since oxygen exchange between the blood and air does not occur here. If the volume of this dead space is V_d, the lungs must draw in a volume V in order to obtain a volume $V-V_d$ of fresh air, which is called the tidal volume. The surface of the pulmonary structure consists of nonciliated moist epithelium that, although devoid of the secretory element found in the tracheobronchial tree, is covered by a surface-active material, without which alveoli would totally collapse (atelectasis) at the end of respiration.

C. FACTORS AFFECTING THE DEPOSITION AND RETENTION OF PARTICULATE MATTER IN THE RESPIRATORY SYSTEM

1. Physical-Mathematical Treatments

The theoretical physical-mathematical treatments use information on the anatomical structure and airflow rates in the respiratory tract, together with knowledge of the physical factors influencing the deposition of particles. The deposition mechanisms will be considered first; aerodynamic factors connected with airflow and gas mixing in the respiratory system will then be mentioned before proceeding to a discussion of the three main models which have been developed.

a. Deposition Mechanisms

Three mechanism are of importance in the deposition of particulate matter in the respiratory tract—inertial impaction, gravitational settling (sedimentation), and diffusion (Brownian motion). The relative significance of these deposition mechanisms varies with anatomical and physiological factors, with the nature of the airflow, and with the characteristics of the aerosol such as particle shape and density.

Particles in an airstream impinge onto a surface when the inertia of the particle is great enough to overcome the resistive forces of the medium and intercept the surface of the obstacle. If gravity is neglected, the path of the particle is determined by the air velocity, the mass and air resistance of the particle, and size and shape of the obstacle. Inertial impaction is therefore of greatest importance in the deposition of large particles of high density, and at points in the respiratory system where the direction of flow changes at branching points in the airways.

The size of the particle as well as its density are significant factors in determining the importance of deposition by gravitational settling. The terminal velocities (settling velocities) of spheres of unit density in air for 10-μ, 1-μ, and 0.1-μ particles are 2.9×10^{-1} cm/sec, 3.5×10^{-3} cm/sec, and 8.6×10^{-5} cm/sec respectively. Density also plays a part since the sedmimentation behavior of a 0.5-μ particle of density 10 g/cm^3 would be equivalent to that of a unit density particle of 1.5-μ diameter. Hence, gravitational settling is most important in the depositon of large particles or of high-density particles such as dusts of heavy metals. Irregularly shaped particles will have an aerodynamic size less than the size predicted on the basis of the measured geometric diameter.

The third main mechanism active in bringing about deposition of particles in the respiratory tract is Brownian movement or diffusion; it is the result of bombardment of the particles by air molecules which are in rapid, random, thermal motion. Diffusion is negligible for large particles (say above 0.5-μ diameter), while it may be the major mechanism for the deposition of small particles (below 0.1μ) in the lower pulmonary tract, where airflow rates are lower and the distances to the walls are less.

b. Aerodynamic Factors

Two aerodynamic factors, namely, flow rates and gas mixing, are incorporated in the models of the respiratory system.

During the respiratory cycle, the actual airflow rate varies from zero up to a maximum value and then back down to zero. Usually the expiratory phase is longer than the inspiratory phase and there may be pauses between the two phases. A study of the respiratory airflow patterns of healthy young men at rest and under a wide range of work loads was made by Silverman *et al.*[9] The maximum inspiratory flow rate increased from a mean value of 40 1/min in sedentary subjects to 100 1/min at an exercise level of 622 kg-m/min and to 286 1/min at an exercise level of 1660 kg-m/min. The corresponding values for maximum expiratory flow rates were 32, 107, and 322 1/min. The collected results are shown in Table 9-1.

Changes in flow rate resulting from physical activity have a profound effect on particle deposition in the respiratory system; this effect in turn depends upon the aerodynamic size of the particulate material inhaled. It has been demonstrated by Amdur, Silverman, and Drinker [10] that the inhalation of irritant particulate material, such as sulfuric acid mist, can decrease the maximum inspiratory and expiratory flow rate in human subjects. Such alterations produced by irritant particles could also affect deposition patterns of particulate matter in general, and may represent the physiological defense response of the body.

Table 9-1.—RESPIRATORY AIRFLOW PATTERNS FOR A GROUP OF HEALTHY YOUNG MEN.[9]

Exercise level	Inspiratory flow rate 1/min (max)	Expiratory flow rate 1/min (max)
Sedentary	40	32
622 kg-m/min	100	107
1660 kg-m/min	286	322

Another factor to be considered in the deposition of particulate matter in the lung is the role of mixing of intrapulmonary gas flow. A study of this factor has been made by Altshuler and co-workers [11] using 0.4-μ particles suspended in air. From the measured wash-in and wash-out rates (after return to particle-free air), the authors were able to calculate the volume of new air which mixed with the residual air. Their data showed that at a tidal volume of 500 ml, not more than 11 percent to 27 percent of new air in each successive breath actually mixed with the residual air. It follows that nondiffusible particles (above 0.5 μ) will tend to penetrate only to the depth of the new air, while smaller particles will have great enough diffusion velocities to move independently into the static air in the lung, in the same way that gas molecules do.

c. *The Models*

For the present purposes, the most important model is that of the Task Group on Lung Dynamics.[4] However, two earlier studies, those of Findeisen [12] and Landahl,[13] are of significance, since they were both considered as a basis for the Task Group model. The Task Group finally selected Findeisen's anatomical model, since it appeared that the rather more sophisticated treatment of Landahl gave no better estimates of deposition values. Further, the cumulative volume down to the end of the terminal bronchioles in the Findeisen model is more in keeping with the anatomical dead space as determined by physiological measurements.

Findeisen [12] predicted the percentage of deposition and the site of deposition of particles of various sizes in the respiratory tract. He estimated there would be a critical particle size of about 0.3–μ to 0.4–μ radius for which a minimal amount of 34 percent would be deposited. This deposition would occur predominantly in the terminal airways and alveoli, thus suggesting that particles of this size should not be dismissed as toxicologically unimportant. Findeisen also estimated that the percentage deposition of 0.3–μ radius particles would be 68 percent as much as that of 1–μ radius particles. His calculated depositions are shown in Figure 9–3. Experimental work on deposition of particles smaller than 1–μ diameter have borne out Findeisen's predictions.

Findeisen's concepts of the anatomy of the pulmonary tract have been criticized by Weibel.[14] Findeisen assumed equal and constant flow rates for inspiration and expiration, a situation which does not prevail during the respiratory cycle. Furthermore, this simple respiratory pattern does not take into account the unique distribution of air in the lungs, or the factors of intrapulmonary mixing of tidal and residual air in the lungs.

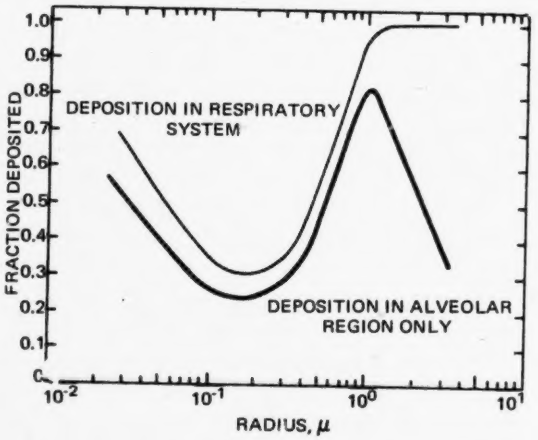

FIGURE 9–3. Calculated Fraction of Particles Deposited in the Respiratory Tract as a Function of Particle Radius.[13] (The figure represents the calculated efficiencies of deposition of particles of various sizes, in the tracheobronchial and alveolar regions of the respiratory system, and shows the size for minimum efficiency.)

Landahl [13] made a similar theoretical study of the problem of particle deposition in the respiratory tract, but included the mouth and pharynx in his anatomical model. He employed several tidal volumes and breathing frequencies in his calculations, but in all cases assumed a constant flow for both the inspiratory and expiratory phases. The calculations yielded deposition values for both phases and took into account the progressively smaller fraction of each tidal volume which penetrated to the various depths. Deposition in various regions for spheres of unit density in the size range 20 μ to 0.2 μ was predicted to fall to a minimum at the size where the predominant force bringing about deposition is shifting from gravitational settling to diffusion. An increase was found in retention with larger tidal volumes.

Landahl [15] also made a separate consideration of deposition in the nasal passages. Earlier studies did not take into account impaction of particles on nasal hairs, or deposition by inertial forces as the airflow changes direction, or sedimentation within the nasal chamber. Assuming a flow rate of 18 1/min, about 75 percent of 10–μ particles would be retained in the nose. Corresponding values would be about 50 percent for 5–μ particles and 10 percent for 1–μ particles. Landahl calculated that at flow rates greater than 18 1/min, essentially 100 percent of the particles above 10 μ would be retained in the nose and that there also would be substantial retention of 2–μ to 5–μ particles. Nasal deposition is negligible for particles below 1 μ in diameter. It follows that the estimated pulmonary deposition values of Findeisen would have to be revised downward for the large particles.

The Task Group on Lung Dynamics used the conventional division of the respiratory tract into three compartments, and made three fundamental assumptions in the development of their model. These were:

1. The log-normal (Chapter 1–B) frequency distribution is generally applicable to particle sizes in the atmosphere. It should be noted that this assumption is by no means universally accepted.
2. The physical activity of the individual affects deposition primarily by its action on ventilation, since physiological adjustment to the demands of increased minute ventilation is to increase tidal volume more than respiratory frequency. In terms of producing the greatest change in deposition throughout the respiratory tract, the effect of an increase in volume at a constant respiratory frequency was considered. (A frequency of 15 breaths per minute was used together with tidal volumes of 750, 1450, and 2150 cm^3 (BTPS); the lowest tidal volume is considered representative of a mild-to-moderate activity state.)
3. The aerodynamic properties of the particle, the physiology of respiration, and the anatomy of the respiratory tract provide a basis for a meaningful and reliable deposition model.

The Task Group's aim was to amalgamate size-deposition relationships into a generalized form which would directly permit the prophetic use of dust-sampling information. By conventional sampling methods, the count median diameter or the mass median diameter is obtainable along with the geometric standard deviation, σ_g (Chapter 1–A). The measurement of one diameter permits the calculation of the other, and both can be expressed as aerodynamic (Stokes) equivalent diameters. The Task Group used calculations basically similar to those of Findeisen [12] to estimate the deposition in the three respiratory compartments. When the mass median aerodynamic diameters of 0.01 μ to 100 μ of various distributions, as represented by different geometric standard deviations, were plotted against the estimated mass deposition of particles occurring in each respiratory compartment, surprisingly little variability was seen with σ_g ranging from 1.2 to 4.5. The results of these calculations are shown in Figure 9–4; the curves indicate a relationship between the mass median aerodynamic diameter and the gravimetric fraction of the inhaled particles which would be deposited in each anatomical compartment. Within these limits (0.01 μ to 100 μ), the mass median aerodynamic diameter served

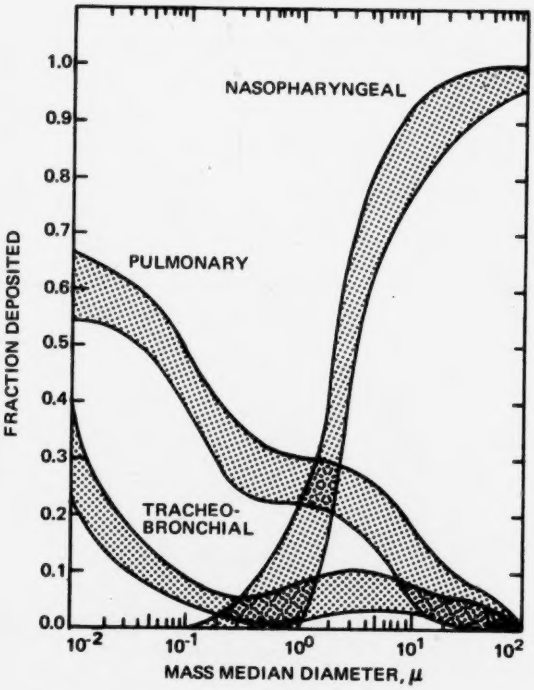

Figure 9–4. Fraction of Particles Deposited in the Three Respiratory Tract Compartments as a Function of Particle Diameter.[4] (This figure shows the deposition efficiencies calculated by the Task Group on Lung Dynamics.)

to characterize the deposition probabilities of the entire particle size distribution from which it emerged. Other parametric functions of the particle distribution failed to produce such simple cohesive relationships.

The effect of the varying tidal volumes is shown in the original report (Figure 12, Reference 4), but it is possible, for practical purposes, to use a mean curve for 1450 ml to represent all three respiration states. This implies that the minute volume will control the total amount of particulate material deposited but will not ordinarily have much effect on the percentage deposition.

The final step was to plot the combination of mean distribution versus mean respiratory performance curves on log-probability paper, a manipulation suggested by the sigmoid shape of the nasopharyngeal and pulmonary deposition curves. The results are shown in Figure 9–5 and Figure 9–6. The deposition in the tracheobronchial compartment is considered, for practical purposes, as constant at approximately 8 percent of the inspired particulate matter.

The results of the Task Force's calculations suggest the type of atmospheric sampling data that will be most meaningful in the correlation of atmospheric particle concentrations and human health. In particular, the insensitivity of the model to aerodynamic size distribution leads the Task Force to propose the concept of "respirable" dust samples, in which the samplers are designed and calibrated to provide data from which one can determine the mass median diameter in aerodynamic terms. Such considerations have a special significance in connection with particles, such as asbestos, which possess an abnormal deposition behavior.

2. Experimental Studies of Factors Affecting Deposition and Retention

Experimental studies of the deposition of inhaled particulate material may be divided into two broad categories. The first group

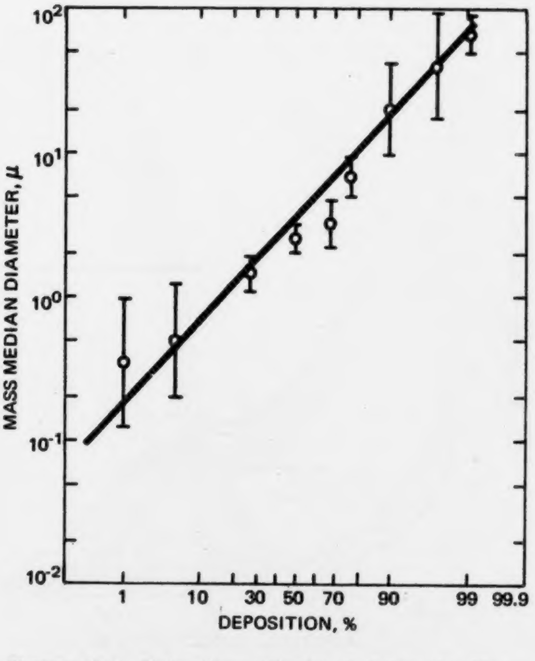

FIGURE 9–5. Data from Figure 9–4 for the Nasopharyngeal Compartment Plotted as a Log-probability Function. (Minute volume: 20 1/min). (The figure shows that there is a roughly linear relationship between deposition efficiency and the logarithm of the particle size.)

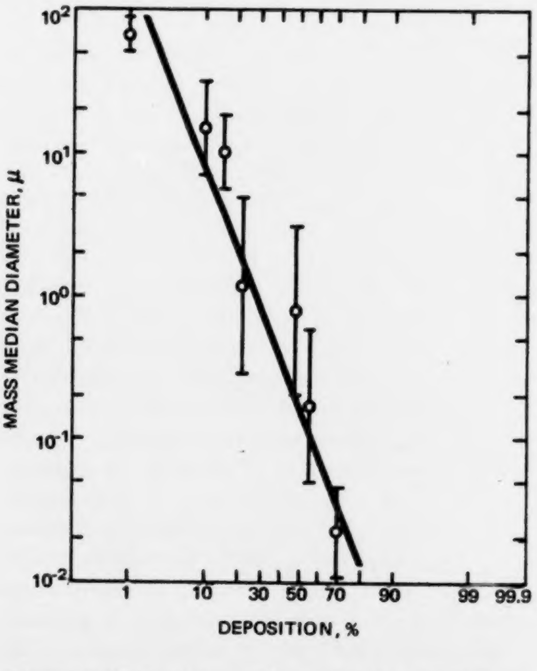

FIGURE 9–6. Data from Figure 9–4 for the Pulmonary Compartment Plotted as a Log-probability Function (Minute Volume: 20 1/min). (This figure shows that there is a roughly inverse linear relationship between deposition efficiency and the logarithm of particle size.)

deals with the measurement of total deposition in the respiratory tract, and the second group is concerned with regional deposition within the various areas of the respiratory tract.

a. Total Deposition in the Respiratory Tract

Experimental studies of deposition were first made by Lehmann *et al.*,[16] Saito,[17] Owens,[18] Baumberger,[19] and Sayers *et al.*[20] Drinker *et al.*,[21] measured respiratory deposition in man with simultaneous recording of respiratory frequency and minute volume. The concentration of particulate matter was measured in the chamber, from which air was inhaled, and in the exhaled air. An average retention value of 55 percent at a frequency of six to 18 respirations per minute was found.

(1) *Effect of Particle Size.*—The first systematic study of the influence of particle size on the percentage deposition of inhaled dust was made in 1940 by Van Wijk and Patterson.[22] The subjects at rest, inspired mineral dust particles from the air of South African gold mines. Percentage deposition approached 100 percent above 5 μ and had decreased to about 25 percent at 0.25 μ, to the limit of the measuring technique.

Brown [23, 24] made a series of tests on human subjects in which he found that the percentage deposition is directly proportional to the particle size and to the density of the suspended material. (Size refers here to the physical dimensions of aggregates of particles rather than to unitary particles.)

More sophisticated experiments by Altshuler *et al.*[25] have used a homogeneous aerosol of triphenyl phosphate, with particle sizes in the range 0.14 μ to 3.2 μ to study deposition in human subjects. It was found that deposition was dependent on particle size and that the minimum deposition diameter was 0.4 μ, which is in reasonable accord with the prediction made by Findeisen (0.6 μ to 0.8 μ) 20 years earlier.

The deposition of coal dust in human subjects has been shown [26] to rise from a minimum efficiency of about 30 percent at 0.5 μ to almost 60 percent at 0.1 μ (mass median diameter). Such an overall efficiency suggests that the absolute efficiency of deposition of the particles smaller than 0.1 μ in the pulmonary air spaces is close to 100 percent. Direct measurement of the particulate matter concentration in samples of alveolar air showed an efficiency of removal of essentially 100 percent down to about 0.5 μ and better than 80 percent for particles well below 0.1 μ.[27]

A hygroscopic particle may collect sufficient water to increase its size significantly over that in the dry state. Dautrebande and Walkenhorst [26] have therefore compared the deposition of sodium chloride with that of coal dust. Using the dry size of the salt particles, deposition curves different from those for coal dust were obtained, but on correcting (by a factor of seven) to account for the growth of salt to liquid droplets in the respiratory tract, the curves were quite similar for the two particles. This result is of considerable practical significance, as it means that hygroscopic particulate air pollutants will be deposited to a higher degree than mineral particles of an equal size. The Task Force Report [4] gives equations which may be applied to correct for the effect of hygroscopicity on deposition.

(2) *Physiological parameters.*—In Brown's experiments,[23, 24] the effect of varying respiratory frequency, tidal volume, and minute volume was determined by making measurements on subjects breathing at rest, under various work loads on a bicycle ergometer, and breathing air containing added CO_2. The conclusions of this work were:

1. Percentage deposition is inversely proportional to respiratory rate for rates below 20 per minute and an increase in frequency above 30 per minute causes no further change in percentage deposition;
2. Percentage deposition is inversely proportional to the minute volume; and
3. Percentage deposition is unaffected by tidal volume, vital capacity, or relative humidity of the inspired air.

The results are essentially in agreement with the predictions of Findeisen and Landahl (although no effect of tidal volume on deposition was observed).

In general agreement with Brown's result for respiratory rate, Altshuler *et al.*[25] found in their experiments that slower, deeper breathing gave greater deposition than faster, shallow breathing. It is also shown that, in agreement with prediction, the differences due to respiratory frequency are greater for 1.6-μ particles than for 0.14-μ particles. For the 1.6-μ particles, impaction and settling are the dominant mechanisms of deposition, and the number settling varies as the first power of time. With the 0.14-μ particles, Brownian motion is the dominant deposition mechanism, and the number settling varies as the square root of time.

Experiments with stearic acid particles [28] have revealed an interesting phenomenon of minimal deposition at normal breathing frequencies of 15 to 20 breaths per minute and an increase when the frequencies were either higher or lower than this. A range of respiratory rates from less than 5 per minute to more than 35 per minute was used, and particle diameters lay between 1 μ and 5 μ. On the other hand, Morrow and Gibb [29] find that the deposition of 0.04-μ sodium chloride particles in dogs and human subjects decreases with an increase in breathing frequency. The deposition percentages themselves (66.5 percent in dogs, 63.4 percent in man) are close to those predicted by Findeisen. An increase in tidal volume increased the deposition in these experiments in distinction to the absence of a tidal volume effect found by Brown.

There appears to be a direct relationship between percentage deposition and holding time in the lungs for particles of 0.55 μ diameter.[30]

b. Regional Deposition

The experimental study of regional deposition of particles is more complex than is the determination of overall total deposition in the respiratory tract. Various specialized techniques such as the inhalation of radioactive particles followed by external counting over specified portions of the chest, or radioautography of the lungs, have been employed. The technique of fractionating exhaled air and counting the particles in each fraction has also been used.

(1) *Nasal Fractionation.* — Lehmann,[31] using a test dust of unspecified size, found a median nasal deposition of 46 percent in 185 normal subjects and 27 percent in 241 silicotics. Dust-laden air was blown through the nose and out through a tube in the mouth. Tourangeau and Drinker [32] found deposition efficiencies of 10 percent to 25 percent with airflow rates through the nose of 4 to 12 liters per minute. Their dust was calcium carbonate of a size comparable to the silica particles found in silicotic lungs. Reversal of the direction of flow did not alter the values.

The most extensive experimental studies of nasal penetration have been made by Landahl's group.[33, 34] The results, especially for corn oil particles, confirm theoretical predictions,[15] and nasal deposition is found experimentally to have a strong dependence on airflow rate.

(2) *Lung Deposition.*—Wilson and La Mer [35] used an aerosol of glycerol containing Na^{24}Cl as a tracer in seven normal subjects who breathed through the mouth at varying frequencies. Particle sizes were in the range of 0.2 μ to 2.5 μ. External chest counts enabled estimates of lung burden to be made, and the pulmonary deposition curve showed a maximum of about 80 percent for particles of about 1.6-μ diameter. A second peak at 0.4 μ was interpreted in terms of the differing optimum sizes for deposition in the finest airways and in the pulmonary air spaces; deposition in these two areas was not distinguished by the method employed.

Indirect estimates of upper respiratory, alveolar, and total deposition in human subjects were made by Brown *et al.*,[36] using a technique for fractionating the exhaled air. In each successive portion, the CO$_2$ content was measured together with the particle count and thus the amount of lung air in each portion of the sample could be estimated. Equal removal efficiencies in both directions were assumed, and a series of expressions was developed for calculating total, alveolar, and upper respiratory deposition. The median particle size of the china clay test dust ranged from 0.24 μ to above 5 μ, and the total retention decreased systematically from 90 percent or more for particles 5 μ and greater, down to 25 percent to 30 percent for 0.25-μ particles. Tracheobronchial retention also decreased systematically with particle size but reached zero at a finite size above 1 μ. Alveolar retention, calculated as the percentage of the number of particles reaching the alveoli, remained between 90 percent and 100 percent for all sizes down to about 1 μ; below that, it decreased in proportion to total retention. The calculated curve for alveolar deposition showed an optimum size at 1 μ.

(3) *Comparative Human and Animal Retention.*—A technique essentially similar to that used by Brown in his experiments on human subjects (see Section C-2-a-(2) above) has been employed by Palm, McNerney, and Hatch [37] for studies on guinea pigs and monkeys. This provides a valuable chance to compare results of experiments on laboratory animals and man. The test dusts included china clay, carbon, antimony trioxide, and *bacillus subtilis* var. niger. The overall pattern of the results was similar in the experimental animals and in man. The actual deposition and retention values were very close for the monkey and for man. For the guinea pig, total retention was close to 100 percent for 3-μ particles and fell systematically with decreasing particle size. In comparison with man, the total retention was higher, especially in the lower size range. Alveolar retention was essentially the same in both species, and it is the tracheobronchial retention which is higher in the guinea pig than in man. Although alveolar deposition in the guinea pig was much lower for 1.5-μ particles than in man, because of the removal of these particles in the tracheobronchial tract, the optimum size for alveolar deposition was similar in both species.

3. Conclusions Reached about Alveolar Deposition

Three important conclusions may be reached about the effect of particle size on alveolar deposition:

1. There is a maximum efficiency of deposition at a size between about 1 μ and 2 μ;
2. There is minimum efficiency for a size of around 0.5 μ; and
3. The percentage of particle deposition for sizes less than 0.1 μ is just as great as for sizes more than 1 μ (Figure 9–3). This last conclusion is not always given enough weight, even though its prediction by Findeisen [12] has been adequately confirmed experimentally.[26, 29]

On the other hand, the importance of the second conclusion above should not be overemphasized, as it refers only to the probability of deposition. If their number, and therefore the mass deposited, is relatively great (as may well be for aged aerosols), then particles in the 0.1-μ to 0.5-μ size region may be as important as smaller and larger particles in provoking toxic response. This is also important when considering particles containing absorbed material.

D. FACTORS AFFECTING THE CLEARANCE OF PARTICULATE MATTER FROM THE RESPIRATORY SYSTEM

A well-known response of a living organism to foreign matter is its attempt to rid itself of, or in some way inactivate, the unwanted material. The overall effectiveness of clearance mechanisms in the lung is well illustrated by the finding that the actual amount of mineral dust found in the lungs of miners or city dwellers at autopsy is only a minor fraction of the total dust that must have been deposited there during their lives. The clearance of certain particles may be very slow. The rate is dependent upon size, site of deposition, and chemical constitution.

Relative to other factors, the importance of removal from the respiratory system of trapped particulate materials depends on the rate at which the material elicits a pathological or physiological response. The effect of an irritant substance which produces a rapid response may depend more on the amount of initial trapping than on the rate of clearance. On the other hand, materials such as carcinogens, which may produce a harmful effect only after long periods of exposure, may exhibit activity only if the relative rates of clearance and deposition are such that a sufficient concentration of material remains in the body long enough to cause

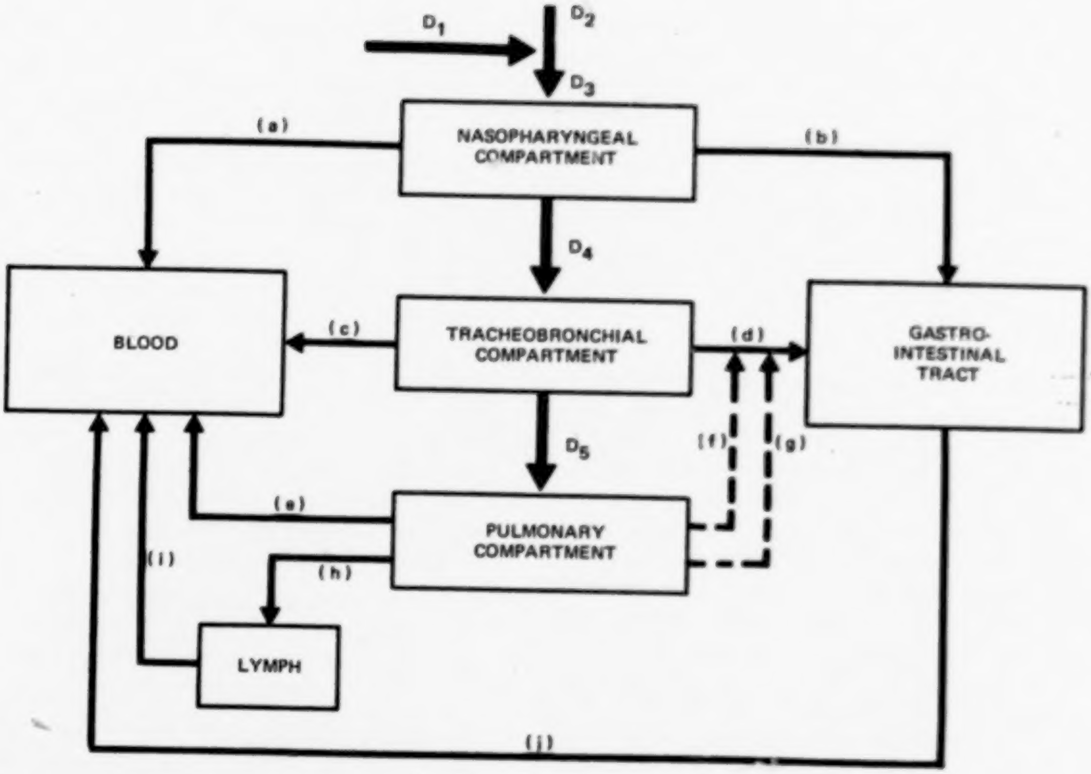

FIGURE 9–7. Schematic Portrayal of Dust Deposition Sites and Clearance Processes. (This diagram illustrates the various deposition sites and clearance mechanisms used in the model of the lung developed by the Task Group on Lung Dynamics, and described in the text.)

pathological change. In such a case, the amount of initial deposition will be of relatively minor importance.

Different clearance mechanisms operate in the different portions of the respiratory tract, so that the rate of clearance of a particle will depend not only on its physical and chemical properties such as shape and size, but also on the site of initial deposition. Furthermore, the presence of a nonparticulate irritant or the coexistence of a disease state in the lungs may interfere with the efficiency of clearance mechanisms and thus prolong the residence time of particulate material in a given area of the respiratory tract. Kotin and Falk[38] have emphasized the possible importance of this interaction in the pathogenesis of lung cancer (Chapter 10).

1. Clearance Model

The Task Group on Lung Dynamics[4] considered the respiratory clearance of particulate matter, as well as its deposition, and the model they developed provides a convenient starting point for a discussion of experimental work on clearance.

Figure 9–7 presents a schematic diagram of all deposition sites and clearance processes. The three conventional compartments of the respiratory tract discussed in connection with the deposition model are used here. D_1 is the particulate material inhaled; D_2 is the material in the exhaled air; D_3, D_4, and D_5 are the particles deposited in the nasopharyngeal, tracheabronchial, and pulmonary compartments respectively, expressed as percentages of D_1 and determinable from the deposition model. In addition to the respiratory tract, three other compartments are listed: The gastrointestinal tract, systemic blood, and the lymph. The different absorption and translocation processes which are associated with the clearance of various compartments are as follows:

a. Rapid uptake of material deposited in the nasopharynx directly into the bloodstream.

b. Rapid clearance of all particulate matter from the nasopharynx by ciliary transport of mucus. This route, and d, have clearance half-times of minutes.

c. Rapid absorption of particles deposited in the tracheobronchial compartment into the systemic circulation.

d. The rapid ciliary clearance of the tracheobronchial compartment. Particles cleared by this route go quantitatively to the gastrointestinal tract.

e. The direct translocation of material from the pulmonary region to the blood.

f. The realtively rapid clearance phase of the pulmonary region dependent on recruitable macrophages. This in turn is coupled to the ciliary mucus transport process for which a half-time of 24 hours has been suggested.

g. A second pulmonary clearance process, much slower than f, but still dependent upon endocytosis and ciliary mucus transport. This process is rate-limited in the pulmonary region by the nature of the particles per se.

h. The slow removal of particles from the pulmonary compartment via the lymphatic system.

i. A secondary pathway in which particles cleared by pathway h are introduced into the systemic blood.

j. The collective absorption of cleared material from the gastrointestinal tract into the blood. No attempt is made to include this factor in the development of the model.

The use of the model requires a knowledge of values for two parameters for each of the pathways just described. These are the amount of material residing in the compartment which follows a particular exit path (regional fraction), and the rate at which that fraction of the material is cleared (biological half-time). In some cases the kinetic values are physiologically controlled and are more or less independent of the nature of the particulate material. In other cases the physiochemical nature of the deposited material is the critical factor.

A classification of inorganic compounds is given in the Task Force Group Report, which contains the best available information on both deposition and clearance of inhaled particles.

The Task Force Group stressed the need for research in the area of solubilities of various compounds in water, in very dilute alkali, and in the presence of proteins. Some of the work done by Morrow *et al.*[39] correlates clearance of material from the lungs with other properties such as ultrafilterability and clearance from intramuscular injection sites. These data will be of value in understanding the effect of physiochemical properties on clearance.

It will be seen that the clearance mechanisms can be divided broadly into those depending on ciliary action and those which operate in the virtually nonciliated pulmonary region. The two sections which follow describe experimental work concerned with these two general subdivisions.

2. Clearance from the Tracheobronchial System

A blanket of mucus in the tracheobronchial region is kept in continual upward movement by the ciliary activity of the columnar epithelium lining which extends down as far as the terminal bronchioles. The frequency of the ciliary beat has been found[40, 41] to be 1,300 per minute in the rat, while the overlying mucous fluid was found to move at an average rate of 13.5 mm per minute. Similar transport rates of 15 mm per minute in excised trachea, and 18 mm per minute in intact animals, have been reported by Antweiler.[42] In this latter study, several particulate materials were used, including soot, coal dust, lycopodium spores, cork dust, aluminum powder, and glass and lead spheres. Transport rates seemed to be unaffected by size, weight, or shape of the particles except in the case of the glass spheres, which were said to be sufficiently smooth to allow the mucus to flow over them rather than to transport them.

It has been demonstrated [43, 44] that in the human lung the mucus flows over only 10 percent to 20 percent of the theoretically available surface at the dividing passages where the airways branch. The mucous blanket divides and flows in two directions around the margins of the opening. This phenomenon, taken together with the greater probability of impact deposition at bifurcations, may explain why histological sections show accumulations of particles at bronchial branch points.[45]

In studies of respiratory system clearance, use has been made of mono-disperse aerosols tagged with radioactive substances to permit the subsequent fate of the material to be followed by external counting techniques. In general, the results indicate that clearance occurs in two distinct phases of about two and ten hours duration, probably representing the clearance of particles from the proximal and distal parts of the bronchial tree. Albert *et al.*[46] studied clearance from the human lung of 3-μ and 5-μ iron oxide particles tagged with 51Cr and 198Au. The rapid phase of clearance was completed within one day, and in most cases within 12 hours. The clearance curves obtained in these studies did not differ substantially from those obtained earlier by Albert and Arnett [47] who used a heterogeneous iron oxide aerosol, suggesting that the actual distribution function of particle sizes may be of little importance in determining clearance rates. However Holma [48] generated a "bi-disperse" aerosol of 6-μ and 3-μ polystyrene particles, tagged respectively with ^{198}Au and ^{46}Sc, and measured simultaneous clearance of the two sizes in rabbits. The larger particles cleared more rapidly than the smaller ones. The mean half-life for the initial phase was 0.48 hours for the 6-μ particles and 1.07 hours for the 3-μ particles. The respective half-lives for the long clearance phase were 69.7 hours and 210 hours.

The effect of irritant gases on clearance of particles is obviously of significance where air pollution involves this combination of factors.

Dalhamn's [40, 41] studies showed that the acute response to irritant gases such as ammonia, formaldehyde, or sulfur dioxide was a cessation of ciliary beat. The time to cessation was dose-related. Chronic exposure of rats to one of the irritants (sulfur dioxide) slowed down or caused a complete cessation of the transport of tracheal mucus, but the average beat frequency of the cilia was the same as in normal animals. The cessation of clearance was a result of an increase in the thickness of the mucous layer of from 5 μ to 25 μ.

The material carried upward by ciliary action is swallowed and thus enters the gastrointestinal tract. Brieger and LaBelle [49] exposed animals to a water-insoluble dye and demonstrated that 24 hours after the termination of exposure, over 50 percent of the total dye found in the body was in the intestinal tract. This phase could persist for several days, during which time a significant intestinal burden was present. The concentration of dye finally fell to very low values after a week or so, and most of the dye that remained in the body was found in the lung, indicating that the rapid phase of ciliary clearance was finished. It has also been shown [50] that uranium dioxide, cleared from the respiratory system to the gastrointestinal tract by ciliary action, is responsible for the high urinary uranium levels seen during the first days after exposure to uranium dioxide dust. The possibility of consequences of relatively high concentrations of an atmospheric pollutant appearing in organs remote from the lungs must not be overlooked. In this connection, the high incidence of stomach cancer in areas suffering from high levels of atmospheric pollution takes on a particularly ominous appearance.

3. Clearance from the Alveolar Surface

Particles deposited on the alveolar surface may be removed by any of the mechanisms (e) to (i) given in the clearance model of Section D–1, and they may also become sequestered by a tissue reaction within the lung (pneumoconiosis), or become bound to protein material in the lungs. Of the several clearance mechanisms, the most rapid is that involving recruitable macrophages (phagocytic cells contained on the alveolar surface epithelium). Phagocytosis may also serve to render the particles incapable of injuring or irritating the alveolar surface epithelium and may to some extent prevent the penetration of the particles into the interstitium of the lung. The origin and behavior of these alveolar macrophages have been the subjects of active research in recent years.[45, 51–53]

LaBelle[54, 55] demonstrated that the clearance of particulate matter by phagocytosis can be markedly influenced by the dust load presented to the lung. The initial observation was made that clearance curves obtained with microgram quantities of activated uranium dioxide were noticeably different from those obtained earlier using milligram amounts. When carbon particles were added to the activated uranium dioxide to bring the total weight of administered dust into the range of the earlier studies, the clearance curves were quite similar. In seeking the reason for this finding. LaBelle demonstrated that the number of free phagocytes washed out of the lungs was related to the dust load and that the amount of dust eliminated from the lungs during the early postexposure period was proportional to the number of free phagocytic cells present. Within the limits of experimental error, the kinetics of elimination of particles and the kinetics of the disappearance of the phagocytic cells following exposure were identical for both inhalation and intratracheal exposures.

The relationship between concentrations in the different respiratory regions during alveolar clearance has been the subject of a short-term study by Gross, Pfitzer, and Hatch.[56] The clearance of four kinds of dust burdens (antimony trioxide, ferric oxide, quartz, and coesite) was followed in rats whose lungs had been loaded using the inhalation and intratracheal injection techniques. Initially, although the greater concentration of dust was to be found in the proximal alveoli, dust deposition was also prominent in alveoli distal to alveolar ducts. However, within 3 or 4 days, the dust in the distally situated alveoli had largely disappeared and had apparently become concentrated in the proximal alveoli. This stagnation of dust in the evaginating alveoli of the respiratory bronchioles and alveolar ducts may help to explain the greater vulnerability of these regions to inhaled irritants.

E. SUMMARY

The respiratory system may be divided into three sections—nasopharyngeal, tracheobronchial, and pulmonary systems. Deposition and clearance mechanisms may differ for the various parts of the respiratory tract. A particle of any size which passes the nasopharyngeal region may be deposited in the remainder of the respiratory tract and, although the actual mechanism of deposition is primarily dependent upon the particle size, the shape of the particle can also affect the efficiency of its deposition. Consequently, if atmospheric dust loads are to be related quantitatively to health hazards, the dust samplers used for monitoring should have collection characteristics similar or the same as the human lung. The fast phases of the lung clearance mechanisms are different in ciliated and nonciliated regions. In ciliated regions, a flow of mucus transports the particles to the entrance of the gastrointestinal tract, while in the nonciliated pulmonary region phagocytosis by macrophages can transfer particles to the ciliated region. The rate of clearance is an important factor in determining toxic responses, especially for slow-acting toxicants such as carcinogens. In addition, since the clearance of particles from the respiratory system primarily leads to their entrance into the gastrointestinal system, organs remote from the deposition site may be affected. The models developed by the Task Group on Lung Dynamics are used in this chapter as a basis for discussion of experimental data on the deposition, retention, and clearance of particles. These models provide a useful representation of the deposition and clearance mechanisms and have been shown to yield predictions which have often been substantiated by experimental findings.

F. REFERENCES

1. Hatch, T. F. and Gross, P. "Pulmonary Deposition and Retention of Inhaled Aerosols." Academic Press, New York, 1964.
2. Morrow, P. E. "Some Physical and Physiological Factors Controlling the Fate of Inhaled Sub-

stances. I. Deposition." Health Physics, Vol. 2, pp. 366–378, 1960.

3. Davies, C. N. "Inhalation Risk and Particle Size in Dust and Mist." Brit. J. Ind. Med., Vol. 6, p. 245, 1949.
4. "Deposition and Retention Models for Internal Dosimetry of the Human Respiratory Tract." Task Group on Lung Dynamics. Health Physics, Vol. 12, pp. 173-207, 1966.
5. Miller, W. S. "The Lung." 2nd edition. Thomas, Springfield, Illinois, 1947.
6. Krahl, V. E. "Microscopic Anatomy of the Lungs." Am. Rev. Respirat. Diseases, Vol. 80, pp. 24–44, 1959.
7. Davies, C. N. "A Formalized Anatomy of the Human Respiratory Tract." In: Inhaled Particles and Vapours, Vol. 1, C. N. Davies (ed.), Pergamon Press, London, 1961, pp. 81–91.
8. Mitchell, R. I. and Tomashefski, J. F. "Aerosols and the Respiratory Tract." Battelle Techn. Rev., Vol. 13, pp. 3–8, 1964.
9. Silverman, L., Lee, G., Plotkin, T., Sawyers, L. A., and Yancey, A. R. "Air Flow Measurements on Human Subjects with and without Respiratory Resistance at Several Work Rates." Arch. Ind. Hyg., Vol. 3, pp. 461–478, 1951.
10. Amdur, M. O., Silverman, L., and Drinker, P. "Inhalation of Sulfuric Acid Mist by Human Subjects." Arch. Ind. Hyg., Vol. 6, pp. 305–313, 1952.
11. Altshuler, B., Palmes, E. D., Yarmus, L., and Nelson, N. "Intrapulmonary Mixing of Gases Studied with Aerosols." J. Appl. Physiol., Vol. 14, pp. 321–327, 1959.
12. Findeisen, W. Über das Absetzen kleiner in der Luft suspendierten Teilchen in der menschlichen Lunge bei der Atmung." Arch. Ges. Physiol., Vol. 236, pp. 367–379, 1935.
13. Landahl, H. D. "On the Removal of Airborne Droplets by the Human Respiratory Tract. I. The Lung." Bull. Math. Biophys., Vol. 12, pp. 43–56, 1959.
14. Weibel, E. W. "Morphometry of the Human Lung." Academic Press, New York, 1963.
15. Landahl, H. D. "On the Removal of Airborne Droplets by the Human Respiratory Tract. II. The Nasal Passages." Bull. Math. Biophys., Vol. 12, pp. 161–169, 1950.
16. Lehmann, K., Saito, Y., and Girorer, W. "Über die quantitative Absorption von Staub aus der Luft durch den Menschen." Arch. Hyg. Bakteriol., Vol. 75, pp. 152–159, 1912.
17. Saito, Y. "Experimentelle Untersuchen über die quantitative Absorption von Staub durch Tiere bei Genau Beakanntem. Staubgehalt der Luft." Arch. Hyg. Bakteriol., Vol. 75, pp. 134–151, 1912.
18. Owens, J. S. "Dust in Expired Air." Trans. Med. Soc. (London), Vol. 45, pp. 79–90, 1923.
19. Baumberger, J. P. "The Amount of Smoke Produced from Tobacco and the Absorption in Smoking as Determined by Electric Precipitation." J. Pharmacol. Exper. Therap., Vol. 21, pp. 47–57, 1923.
20. Sayers, R. R., Fieldner, A. C., Yant, W. P., Thomas, B. G. H., and McConnel, W. J. "Exhaust Gases from Engines Using Ethyl Gasoline." U.S. Dept. of Interior, Bureau of Mines, Report of Investigations 2661, 1924, 24 pp.
21. Drinker, P., Thompson, R. M., and Finn, J. L. Quantitative Measurements of the Inhalation, Retention, and Exhalation of Dust and Fumes by Man. I. Concentration of 50 to 450 mg per cubic meter." J. Ind. Hyg. Toxicol., Vol. 10, pp. 13–25, 1928.
22. Van Wijk, A. M. and Patterson, H. S. "The Percentage of Particles of Different Size, Removed from Dust Laden Air by Breathing." J. Ind. Hyg. Toxicol., Vol. 22, pp. 31–35, 1940.
23. Brown, C. E. "Quantitative Measurements of the Inhalation, Retention, and Exhalation of Dust and Fumes by Man. II. Concentration below 50 mg per cubic meter." J. Ind. Hyg. Toxicol., Vol. 13, pp. 285–291, 1931.
24. Brown, C. E. "Quantitative Measurements of the Inhalation, Retention, and Exhalation of Dust and Fumes by Man. III. Factors Involved in the Retention of Inhaled Dusts and Fumes by Man." J. Ind. Hyg. Toxicol., Vol. 13, pp. 293–313, 1931.
25. Altshuler, B., Yarmus, L., Palmes, E. D., and Nelson, N. "Aerosol Deposition in the Human Respiratory Tract. I. Experimental Procedures and Total Deposition." Arch Ind. Health, Vol. 15, pp. 293–303, 1957.
26. Dautrebande, L. and Walkenhorst, W. "Über die Retention von Kochsaltteilchen in den Atemagen." In: Inhaled Particles and Vapours, Vol. 1, C. N. Davies (ed.), Pergamon Press, London, 1961, pp. 110–121.
27. Dautrebande, L., Bechmann, H., and Walkenhorst, W. "Lung Deposition of Fine Dust Particles." Arch. Ind. Health, Vol. 16, pp. 179–187, 1957.
28. Dennis, W. L. "Discussion of Davies." In: Inhaled Particles and Vapours, Vol. 1, C. N. Davies (ed.), Pergamon Press, London, 1961, pp. 88–91.
29. Morrow, P. E. and Gibb, F. R. "The Deposition of a Submicronic Aerosol in the Respiratory Tract of Dogs." Am. Ind. Hyg. Assoc. J., Vol. 19, pp. 196–200, 1958.
30. Landahl, H. D., Tracewell, T. N., and Lassen, W. H. "Retention of Airborne Particulates in the Human Lung." III. Arch Ind. Hyg., Vol. 6, pp. 508–511, 1952.
31. Lehmann, C. "The Dust Filtering Efficiency of the Human Nose and its Significance in the Causation of Silicosis." J. Ind. Hyg. Toxicol., Vol. 17, pp. 37–40, 1935.
32. Tourangeau, F. J. and Drinker, P. "Dust Filtering Efficiency of the Human Nose." J. Ind. Hyg. Toxicol., Vol. 29, pp. 53–57, 1937.

33. Landahl, H. D. and Black, S. "Penetration of Airborne Particulates through the Human Nose." J. Ind. Hyg. Toxicol., Vol. 29, pp. 269–277, 1947.
34. Landahl, H. D. and Tracewell, T. N. "Penetration of Airborne Particulates through the Human Nose." II. J. Ind. Hyg. Toxicol., Vol. 31, pp. 55–59, 1949.
35. Wilson, I. B. and LeMer, V. K. "The Retention of Aerosol Particles in the Human Respiratory Tract as a Function of Particles Radius." J. Ind. Hyg. Toxicol., Vol. 30, pp. 265–280, 1948.
36. Brown, J. H., Cook, K. M., Ney, F. G., and Hatch, T. F. "Influence of Particle Size upon the Retention of Particulate Matter in the Human Lung." Am. J. Public Health, Vol. 40, pp. 450–458, 1950.
37. Palm, P. E., McNerney, J., and Hatch, T. F. "Respiratory Dust Retention in Small Animals. A Comparison with Man." Arch. Ind. Health, Vol. 13, pp. 355–365, 1965.
38. Kotin, P. and Falk, H. "Atmospheric Factors in Pathogenesis of Lung Cancer." Advan. Cancer Res., Vol. 7, pp. 475–514, 1963.
39. Morrow, P. E., Gibb, F. R., Davies, C. N., and Fisher, M. "Dust Removal from the Lung Parenchyma: An Investigation of Clearance Simulants." Toxicol. Appl. Pharmacol. Vol. 12, pp. 372–396, 1968.
40. Dalhamn, T. "Mucous Flow and Ciliary Activity in the Trachea of Healthy Rats and Rats Exposed to Respiratory Irritant Gases." Acta Physiol. Scand., Suppl. 123, 1956.
41. Dalhamn, T. "A Method for Studying the Effect of Gases and Dusts on Ciliary Activity in Living Animals." In: Inhaled Particles and Vapours, Vol. 1, C. N. Davies (ed.), Pergamon Press, London, 1961, pp. 315–317.
42. Antwiler, H. "Über die Funktion des Flimmerepithels der Luftwege inbesondere unter Staubbelastung." Beitr. Silkose-Forsch., Sonderband, Vol. 3, p. 509, 1958.
43. Hilding, A. C. "On Cigarette Smoking, Bronchial Carcinoma, and Ciliary Action. III. Accumulation of Cigarette Tar upon Artificially Produced Deciliated Islands in the Respiratory Epithelium." Ann. Otol. Phinol. Laryngol., Vol. 65, pp. 116–130, 1956.
44. Hilding, A. C. "Ciliary Streaming in the Bronchial Tree and the Time Elements in Carcinogenesis." New Engl. J. Med., Vol. 256, pp. 634–640, 1957.
45. Casarette, L. J. "Autoradiographic Observations after Inhalation of Polonium^{210} in Rats." Radiation Res. (Suppl.), Vol. 5, pp. 187–204, 1964.
46. Albert, R. E., Lippman, M., Spiegelman, J., Strethlow, C., Briscoe, W., Wolfson, P., and Nelson, N. "The Clearance of Radioactive Particles from the Human Lung." In: Inhaled Particles and Vapours, Vol. II, C. N. Davies (ed.), Pergamon Press, London, 1967, pp. 361–378.
47. Albert, R. E. and Arnett, L. C. "Clearance of Radioactive Dust from the Lung." Arch. Ind. Health, Vol. 12, pp. 99–106, 1955.
48. Holma, B. "Short Term Lung Clearance in Rabbits Exposed to a Radioactive Bi-disperse (6 and 3) Polystyrene Aerosol." In: Inhaled Particles and Vapours, Vol. II, C. N. Davies (ed.), Pergamon Press, London, 1967, pp. 189–203.
49. Brieger, H. and LaBelle, C. W. "The Fate of Inhaled Particulates in the Early Postexposure Period." Arch. Ind. Health, Vol. 19, pp. 510–515, 1959.
50. Downs, W. L. "Excretion of Uranium by Rats Following Inhalation of Uranium Dioxide." Health Physics, Vol. 13, pp. 445–453, 1967.
51. Casarett, L. J. "Some Physical and Physiological Factors Controlling the Fate of Inhaled Substances. II. Retention." Health Physics, Vol. 2, pp. 379–386, 1960.
52. Casarett, L. J. and Milley, P. S. "Alveolar Reactivity Following Inhalation of Particles." Health Physics, Vol. 10, pp. 1003–1011, 1964.
53. Policard, A., Collet, A., and Pregermain, S. "Structures alveolaires normales du pneumon examines au microscope electronique." Semaine. Hop. (Paris), Vol. 33, p. 385, 1957.
54. LaBelle, C. W. and Brieger, H. "Synergistic Effects of Aerosols. II. Effects on Rate of Clearance from the Lung." Arch. Ind. Health, Vol. 20, pp. 100–105, 1959.
55. LaBelle, C. W. "Patterns and Mechanisms in the Elimination of Dust from the Lung." In: Inhaled Particles and Vapours, Vol. 1, C. N. Davies (ed.), Pergamon Press, London, 1961, pp. 356–368.
56. Gross, P., Pfitzer, E. A., and Hatch, T. F. "Alveolar Clearance: Its Relation to Lesions of the Respiratory Bronchiole." Am. Rev. Respirat. Diseases, Vol. 94, pp. 10–19, 1966.

Chapter 10

TOXICOLOGICAL STUDIES OF ATMOSPHERIC PARTICULATE MATTER

Table of Contents

Page
A. INTRODUCTION 129
B. MECHANISMS OF TOXICOLOGICAL ACTION OF PARTICULATE MATTER 129
  1. Intrinsic Toxicity 129
  2. Absorbed Substances 130
  3. Reduction of the Toxicity of Irritant Gases 130
C. TOXICOLOGICAL STUDIES OF SPECIFIC PARTICULATE MATERIALS 131
  1. Pathological Studies of Smoke and Carbon Particles 131
  2. Physiological Studies of Response to Particulate Material 132
  3. Experimental Studies of Mixtures of Irritant Gases and Particulate Material 134
D. CARCINOGENESIS 137
  1. Carcinogens 137
  2. Polynuclear Aromatic Hydrocarbons as Carcinogens in Polluted Atmospheres 138
  3. Pathology of Carcinogenesis 141
E. SUMMARY 141
F. REFERENCES 142

List of Tables

Table
10–1 Effect of Exposure of Rabbits to 2 ppm Ozonized Gasoline on Retention of Inhaled Soot 134
10–2 Benzo (a) pyrene Concentrations in Several Urban and Nonurban Areas 138
10–3 Benzo (a) pyrene as a Fraction of the Total Aromatic Hydrocarbon Content of Several Urban Atmospheres 138
10–4 Effect of Various Conditions of Exposure on the Destruction of Some Polynuclear Aromatic Hydrocarbons 139
10–5 Percentage Recovery of Polynuclear Aromatic Hydrocarbons from 0.5μ Soot Particles and from Plasma after Incubation with Plasma for Varying Periods 140
10–6 Distribution of Lung Cancer by Site of Origin 141

Chapter 10

TOXICOLOGICAL STUDIES OF ATMOSPHERIC PARTICULATE MATTER

A. INTRODUCTION

Experimental toxicology, using specific atmospheric pollutants, would be the best means for deriving air quality criteria, provided that man could be used as the experimental animal. However, the ethical impossibility of performing experiments using human exposures to varying concentrations of a wide range of compounds precludes so direct an approach. Although a limited amount of intentional human experimentation may be possible, most of the data for human toxocology are derived from accidental or occupational exposures. The use of laboratory animals in toxicological experiments is more straightforward, but the obvious anatomical and metabolic differences between the animals and man require the exercise of considerable caution in applying the results of animal exposures to human health criteria. Furthermore, many of the animal experiments performed have left something to be desired in terms of air pollution toxicology, since the "end-point" of the experiments has frequently been the death of the animals at exposures to concentrations far in excess of those likely to be found, or tolerated, in the atmosphere. There is a great need for chronic inhalation studies to define long-term effects occurring over a lifespan.

The difficulties and limitations of toxicological studies discussed in the last paragraph should not obscure the fact that an increasing amount of data useful for air quality criteria is being amassed; it is, however, always essential to bear in mind the limitations of the data. Toxicological studies have shown that atmospheric particles may elicit a pathological or physiological response in at least three ways. First, the particle may be intrinsically toxic; second, the presence of an "inert" particle in the respiratory tract may interfere with the clearance of other airborne toxic materials; and third, the particle may act as a carrier of toxic material. There is also evidence that the presence of particles may occasionally reduce the toxicity of a second pollutant; this phenomenon is described briefly. From an air pollution standpoint, one of the most ubiquitous particulate pollutants is smoke; some pathological studies involving this material are presented. Studies of physiological response to irritant particles and to mixtures of particulate matter with irritant gases are described.

A final section of this chapter then deals with carcinogenesis and atmospheric pollutants.

B. MECHANISMS OF TOXICOLOGICAL ACTION OF PARTICULATE MATTER

1. Intrinsic Toxicity

Few common atmospheric particulate pollutants appear to be intrinsically toxic; of these, the most important toxic aerosol is sulfur trioxide (SO_3) (either as the free oxide, or hydrated as sulfuric acid—H_2SO_4), which has a high degree of toxicity, at least for the guinea pig. Although silica (from fly ash) is frequently present as a pollutant, atmospheric concentrations are normally too low to lead to silicosis. In recent years, however, concern has been expressed over a number of less common toxic particulate pollutants, including lead, beryllium, and asbestos. Increasing amounts of lead (as oxides and salts) are being discharged into the atmosphere as a result of the burning of gasoline containing lead additives; on the other hand, other sources contributing to atmospheric lead seem to be decreasing and, according to Stokinger and Coffin,[1] toxic effects due to lead do not seem to present a serious risk to the population at large. Beryllium (as the oxide BeO) can lead to chronic pulmonary disease with a high fatality rate. Accidental exposures have shown that there may be a considerable latency period before disease develops, and some alarm has been generated over the use of beryllium as a rocket fuel, although the rocket effluent is predominantly a "high-fired," relatively inert form of beryllium oxide. The situation with regard to asbestos is also potentially serious. Brief industrial or accidental exposures to asbestos can lead, after a latency period of 40 years or more, to the development of diffuse mesotheliomas of the pleura or peritonium, and it is possible that sufficient levels of asbestos might be generally present in the atmosphere to constitute a definite health hazard. This possibility is made more likely by the greatly increased use of asbestos since the occurrence of the exposures which are only now leading to the development of mesotheliomas. A series of autopsies has shown that an appreciable fraction (20 percent to 50 percent) of the population at large has "asbestos bodies" in its lungs. More detailed discussions of both asbestos[2] and beryllium[3] toxicity may be found in recent reviews.

Several other potential carcinogens are known to be present as relatively minor atmospheric pollutants; these may be particulate in nature, and they are discussed in Section D.

2. Adsorbed Substances

Toxic substances may be adsorbed on the surface of particulate matter, which may then carry the toxic principle into the respiratory system. The presence of carbon or soot as a common particulate pollutant is noteworthy, as carbon is well known as an efficient adsorber of a wide range of organic and inorganic compounds. Some specific studies involving mixtures of particulate matter and irritant gases are presented in Section C–3; the present section is concerned mainly with the general principles of adsorption and desorption.

The role played by the affinity for the adsorbate by the particle is complex. A high affinity will mean that relatively large loads of adsorbate may be carried by each particle. If the adsorbate in its *free* state is slowly removed from the air in the respiratory system, then the deposition of particles carrying high concentrations may constitute a greater toxic hazard, especially at the localized deposition points. Whether or not the effect is significant depends on the efficiency of the desorption and elution process relative to that of the clearance process. The chemical nature of both adsorber and adsorbate, and the size of the adsorbing particle, all play a part in determining these various efficiencies, and each system will show its own individual characteristics. Carcinogens, which may produce their effect only after long or repeated exposure, present a particularly involved situation, because it is not clear whether slow release of small concentrations of the carcinogen is more dangerous than the rapid release of larger quantities. Experimental evidence on the elution of a specific series of carcinogens is described in Section D–2.

3. Reduction of the Toxicity of Irritant Gases

The finding that a preexposure to particulate material will tend to protect animals against the action of an irritant gas is not an uncommon one.

Pattle and Burgess[4] found that, with mice and guinea pigs, the previous inhalation of smoke reduced the toxicity of sulfur dioxide (measured in terms of the dosage required to produce death). They postulated that the reduced toxicity of SO_2 produced by preexposure to smoke was due either to the action of the smoke in reducing the volume breathed (and thus the SO_2 dose received), or to a stimulation of secretions which may protect the mucosa from the irritant action of the gas. This explanation is given weight by their findings that increased toxicity resulted from the administration of mixtures of smoke and sulfur dioxide. A similar study by Salem and Cullumbine[5] indicated that the effect of kerosene smoke on the toxicity of irritant substances depended to some extent on the species of animal, although the toxicity of acrolein and acetaldehyde seemed to be decreased in most cases.

Wagner *et al.*[6] observed that the preexposure of mice to oil mists would protect against the later action of inhaled ozone and nitrogen dioxide. The greatest protection was obtained when the oil mist was given 18 hours prior to the exposure to the irritant gas; when the oil mist and irritant gas were given together, there was an enhancement of toxicity. The relative degree of this enhancement was consistent with the known depth of penetration of nitrogen dioxide and ozone in the respiratory tract and the relative solubility of the two gases in mineral oil. The hypothesis for the protective action of the pretreatment with oil is the formation of an oil film on the aveolar surface.

Again Amdur and Devir,[7] using guinea pigs, have observed that the presence of an aerosol of 2.5μ triphenyl-phosphate particles at a concentration of 50 mg/m^3 to 100 mg/m^3 lessens the increase in pulmonary flow resistance due to inhalation of sulfur dioxide. They then studied the effect of treatment with the particulate material before exposures to sulfur dioxide alone. This sequence afforded excellent protection against the response to the sulfur dioxide, and the degree of protection was related to the total amount of aerosol inhaled.

C. TOXICOLOGICAL STUDIES OF SPECIFIC PARTICULATE MATERIALS

1. Pathological Studies of Smoke and Carbon Particles

Smoke from burning bituminous coal was used for a study on rabbits and rats by Schnurer and Haythorn.[8] Four rabbits and eight rats were exposed for periods of 80 days to smoke with a concentration of 125 million particles/ft^3 (4410 particles/cm^3), of which 8 percent was free SiO_2. Control animals received clean filtered air. One control rabbit and two exposed rabbits died of bronchial pneumonia before the end of the exposure. One rabbit and six rats were autopsied immediately after exposure, and the rest at intervals up to 429 days. Lesions typical of nonoccupational anthracosis were noted in the lungs of the animals killed immediately after exposure, while fibrous reactions developed around the carbon deposits (with the formation of collagen strands) in the animals that were examined several months to a year after the exposure. The authors state that these lung changes were analogous to those seen in a milder grade of bituminous pneumoconiosis of soft-coal miners, and they also concluded that the pneumonitis and fibrosis were attributable to the carbon rather than to the small amount of silica present.

Schnurer[9] has also attempted to compare the response to the burning of equal weights of anthracite coal, coke, and bituminous coal. Unfortunately, widely differing particle concentrations were obtained from burning equal weights of fuel, so that it is impossible to place any simple interpretation on the results of the experiments.

Pattle *et al.*[10] studied the acute toxic effects of smoke, generated by burning tetrahydronaphthalene in the concentration range of 700 mg/m^3 to 1100 mg/m^3. They found that the median dosage to death for guinea pigs and mice lay between 147,000 mg—min/m^3 to 351,000 mg—min/m^3. In mice, the cause of death was blockage of the air passages and delayed death resulting from the exposure was unusual. The guinea pigs showed hemorrhagic lesions, and delayed deaths were more common. The action of smoke on rats resembled that on mice. No data are provided to show that unburnt tetrahydronaphthalene did not enter the smoke stream in these experiments, since toxic effects are well known for this substance.

In the course of a study of mixtures of sulfur dioxide and smoke, to be discussed in Section C–3, Pattle and Burgess[4] reported some data on the effect of smoke alone on mice. The smoke was generated by a burning kerosene lamp, and its concentration was about 50 mg/m^3. The experiment continued for 36 hours followed by a gap of 11 hours. The experiment was subsequently continued for 30 hours. There were no fatalities and none occurred after the experiment. Histological examination of the lungs showed that at the end of exposure the soot particles were partly spread over the lining of the bronchioles and alveoli and partly aggregated into patches. There were no signs of edema, consolidation, hemorrhage, or emphysema and capillary congestion was slight. The lungs were normal except for the presence of carbon particles, and animals killed 3 months after exposure showed phagocytosis and gradual clearance of smoke from the lungs.

Salem and Cullumbine [5] also make brief mention of the exposure of mice and guinea pigs to smoke from a kerosene lamp. The animals survived exposure to 664 mg/m^3 of smoke for 6 hours, and autopsy revealed no obvious damage to the lungs.

The physiological effects on mice of exposure to carbon black by ingestion, skin contact, subcutaneous injection, and inhalation were examined by Nau *et al.*[11–14] Channel black (particle diameter 0.025μ) and furnace black (particle diameter 0.035μ) were used in concentrations of 2.4 mg/m^3 and 1.6 mg/m^3 respectively for the inhalation experiments. Mice, hamsters, guinea pigs, rabbits, and monkeys were exposed for prolonged periods to the dust, but no effect other than the accumulation of carbon particles in the lungs was demonstrated.

The problem of whether exposure of rats to coal dust or smoke would alter their susceptibility to infection by Type I pneumococci was examined by Vintinner and Baetjer [15] as part of a series of studies on the effect of fibrous, inert, and adsorptive dusts on susceptibility to infection in experimental animals.[16–18] The concentration of coal dust varied between 400 and 850 million particles/ft^3 with an average of 700 million particles/ft^3. The smoke level in the chambers would have been considered as "dense" on visual inspection, or equivalent to about a Number 3 reading on the Ringelmann Chart. Analyses of the particulate matter in mg/m^3 in the smoke chamber averaged as follows: Total solids—570, carbon—470, ash—93, silica—46, iron—15, and sulfur—9, that is, about 100 times the values listed as "average composition of susupended dust during the winter months for all cities." The exposures ranged from 5 days to 165 days for the bituminous coal dust and from 2 days to 154 days for the smoke. On the basis of their extensive data, the authors concluded that the inhalation of smoke did not alter the susceptibility to infection when the organisms, either in broth or in mucin, were administered by intrabronchial injection. The bituminous coal dust seemed to exert a slight *protective* effect when the organisms were suspended in mucin but not when they were injected in a broth medium.

Based upon the reported studies, smoke or carbon black on its own apparently produces little major damage to the respiratory system of animals even at exposure levels some orders of magnitude greater than those encountered in polluted atmospheres.

2. Physiological Studies of Response to Particulate Material

Certain particulate materials are pulmonary irritants which have been shown to produce alterations in the mechanical behavior of the lungs, the alteration being predominantly an increase in flow resistance. This was demonstrated by Amdur [19] for sulfuric acid, and by Amdur and Corn [20] for ammonium sulfate, zinc sulfate, and zinc ammonium sulfate, using the guinea pig as an assay animal. (The decreased flow rates observed by Amdur, Silverman, and Drinker [21] in human subjects exposed to sulfuric acid mist probably reflect an increase in flow resistance, although, since the resistance was not measured in those experiments, such a statement cannot be conclusive.) A series of papers making use of the increase in pulmonary flow resistance as an assay tool has been published by Amdur,[22–27] and "response" to an irritant in discussion of her work generally refers to this increase.

Nadel *et al.*[28] report a correlation between the alterations in pulmonary mechanics and actual anatomical change in cats exposed to aerosols of histamine and zinc ammonium sulfate. The authors discuss the physiological mechanisms which may operate to bring about such alterations in pulmonary mechanics.

In connection with a study of the effect of various aerosols on the increase in pulmonary flow resistance in guinea pigs produced by sulfur dioxide, Amdur and Underhill [22] first examined the response to the aerosols alone. These aerosols included spectrographic carbon at 2 mg/m^3 and 8 mg/m^3, activated carbon at 8.7 mg/m^3, manganese dioxide at 9.7 mg/m^3, open hearth dust at 7.0 mg/m^3, iron oxide (Fe_2O_3) at 11.7 mg/m^3 and 21.0 mg/m^3, manganous chloride and ferrous sulfate at

1 mg/m^3, and sodium orthovanadate at 0.7 mg/m^3; the particle sizes were less than 0.5μ. Standard one-hour exposures were used in this routine bioassay method, with the exception of the iron oxide exposures, which were extended to two hours. In no instance did any of the aerosols produce an alteration in flow resistance. The failure of the aerosols to produce an increase in resistance in these experiments suggests that if they cause bronchial constriction, it must be only in extremely high concentrations. From the point of view of air pollution toxicology, none of them would be classed on this basis as pulmonary irritants. An uncharacterized "fly ash" from an oil-fired burner was also tested and proved inert.

At a concentration of 1 mg/m^3, ferric sulfate aerosol produced a 77 percent increase in flow resistance in a group of 15 animals which was statistically significant at the level of $P<0.001$. Ferric sulfate, in distinction to ferrous sulfate, must be classed as an irritant.

On the other hand, Dautrebande and DuBois [29, 30] have reported constriction and increased airway resistance in isolated guinea pig lungs and in human subjects with a wide variety of supposedly "inert" particulate matter. The relationship of their results to Amdur's work is not clear since Dautrebande's particle concentrations appear to be abnormally high. Total lung capacity and vital capacity were not altered by the inhalation of particulate material in healthy subjects, but the vital capacity in patients with pulmonary disease was reduced by the inhalation of particulate materials. It is interesting in this connection that guinea pigs with an initially high pulmonary flow resistance showed a greater response to low concentrations of irritant aerosols or of inert aerosol-irritant gas combinations than animals with average control flow resistance values.[31]

The possibility that the conflict between Amdur's and Dautrebande's work may be resolved in terms of the doses involved is given credence by studies which suggest that human response to coal dust may be dose-related.[32] Coal dust clouds similar to those in mines have been produced in the laboratory, and the number and weight of respirable particles have been measured. Clouds containing 8 mg/m^3, 9 mg/m^3, 19 mg/m^3, 33 mg/m^3, and 50 mg/m^3 dust in the size range of 1μ to 7μ were used. Normal subjects inhaled the dust for 4 hours, and airway resistance was measured with a body plethysmograph. No changes were obtained after the inhalation of coal dust from clouds containing 8 mg/m^3 or 9 mg/m^3; but with concentrations of 19 mg/m^3, 33 mg/m^3, and 50 mg/m^3, significant increases in airway resistance occurred and the response was correlated with the quantity of dust. One hour after exposure ceased, the airway resistance was about two-thirds back to normal. With the two highest concentrations, the respiratory rate increased throughout the 4 hours, and subjects complained of difficulty in breathing after one to two hours. It would appear that if 8 mg/m^3 or 9 mg/m^3 produced no lung function change in a 4-hour period, the material (cold dust) and perhaps other "inert" particulate matter would be unlikely to do so at any concentrations likely to occur in air pollution. In Chapter 1, Table 1–2 shows a maximum geometric mean concentration of urban particulates in 1961 to 1965 of 180 $\mu g/m^3$.

Particle size may play an important part in determining the potency of an irritant. For example, at a mass concentration of 1.4 mg/m^3 to 1.9 mg/m^3, sulfuric acid mist of 0.8μ produces a 51 percent increase in pulmonary flow resistance in guinea pigs as compared to control values,[19] and zinc ammonium sulfate particles of 0.84μ produce a 21 percent increase.[25] If the zinc ammonium sulfate size is 0.3μ the corresponding increase in pulmonary flow resistance is 130 percent.[20] The studies on zinc ammonium sulfate were carried out by Amdur and Corn [20] with nonhomogeneous aerosols with mean sizes by weight of 0.24μ, 0.51μ, 0.74μ, and 1.4μ, at several concentrations. As the particle size decreased over the range 1.4μ to 0.29μ, the response to an equal mass concentration rose. When dose-response curves of percent increase in flow resistance against concentration in mg/m^3 were plotted for the different particle sizes, the slopes increased as the particle size decreased. A similar study was undertaken by Amdur and Creasia [33] using *m*-terphenyl as the aerosol (size range

0.3μ to 2.0μ); this aerosol does not absorb water during passage through the respiratory tract. An essentially, identical pattern to that seen for zinc ammonium sulfate emerged, with the irritant potency increasing with decreasing particle size, and the slopes of the dose-response curves steeper than those for the smaller particles. All of the particles used in these studies fall within the "respirable size range." The possible implications for air pollution are:

1. Particles below 1μ may have greater irritant potency than larger species, and
2. A small increase in concentration could produce a greater-than-linear increase in irritant response when the particles are smaller than 1μ.

The effect of particulate matter on clearance mechanisms has already been mentioned. The deposition of particles affects mucous secretion and ciliary action most markedly at branchings; e.g., miniature ridges throughout the tracheobronchial tree as contrasted with intervening areas, and it is in the ridge areas that alterations in the morphology are initially most intense and prolonged and ultimately irreversible. The adverse effect is manifested by a slowing of the flow of the mucous stream, alteration in the physical and chemical properties of mucus, and changes in ciliary action.

Examination of sections of respiratory epithelium removed from the lungs reveal hyperplastic and metaplastic changes earliest at these sites. The exaggerated effect of the impingement of irritants on respiratory epithelium at branch points taken together with less efficient clearance is consistent with this result. Thus, abnormal retention and accumulation of soot (and possibly carcinogenic particles) may occur, especially in segmental bronchi. The accompanying peribronchial and peribronchiolar inflammatory response further interferes with physiologic mechanisms of defense.

Experimental confirmation of enhanced retention in the presence of irritant material is to be found in the work of Tremer *et al.*,[34] who exposed rabbits first to synthetic smog and then to soot. The results are shown in Table 10–1.

The main conclusions of this section are:

1. The predominant physiological effect of irritants is to increase pulmonary flow resistance;
2. Exceptionally heavy loads of relatively inert particles may cause some increase in flow resistance; and
3. The intensity of physiologic response may increase with a decrease in particle size for any given irritant.

3. Experimental Studies of Mixtures of Irritant Gases and Particulate Material

The possible influence of inert particulate matter on the toxicity of irritant gases has been the subject of considerable speculation[35–37] and a limited amount of experimental work. Such interaction of gaseous and particulate pollutants might be important to understanding the complicated toxicological picture of the air pollution disasters.

Table 10–1.—EFFECT OF EXPOSURE OF RABBITS TO 2 PPM OZONIZED GASOLINE ON RETENTION OF INHALED SOOT.

Specimen	Smog exposure, hours	Room air (recovery), hours	Soot exposure, hours	Room air (recovery), hours	Soot in lung, mg
No recovery period after smog exposure:					
Control	0	0	1	0	3.7
Animal No. 1	1	0	1	0	7.9
Animal No. 2	1	0	1	24	5.4
With varying recovery periods:					
Control	0	0	1	0	1.1
Animal No. 1	1	4	1	0	7.4
Animal No. 2	1	8	1	0	5.2
Animal No. 3	1	24	1	0	2.0

The first experimental evidence that an inert aerosol could alter the response to an irritant gas was presented in 1939,[38] when it was reported that concentrations of mustard gas which were relatively harmless to rats would produce pulmonary edema and death when administered in combination with an inert aerosol (sodium chloride). It was postulated that the adsorption of gas on the particles had increased the amount of irritant vapor reaching the critical target areas of the lung. Dautrebande and his co-workers [39, 40] studied the sensory response of human subjects to pollutants thought to be constituents of the Los Angeles smog and found that the presence of particles of sodium chloride, oil mist, or smoke, increase the irritation of eye, nose, and throat by sulfur dioxide, formaldehyde, and other gaseous pollutants.

LaBelle *et al.*[41] studied the effect of particulate matter on the survival time of mice exposed to formaldehyde, acrolein, and oxides of nitrogen. The particles used included triethylene glycol, ethylene glycol, mineral oil, glycerin, sodium chloride, two commercial filter grades of diatomaceous earth, and a commercial silica gel. From theoretical considerations, the probable percent penetration of the upper respiratory tract by various gases was calculated. If the gas penetrated to a greater extent than the particles, then adsorption on the particles would decrease the amount of irritant gas reaching the lungs and the toxicity would decrease. Such a situation exists with oxides of nitrogen. Conversely, if the gas did not readily penetrate the upper respiratory tract, adsorption on small particles would tend to carry more gas to the lungs and thus increase the toxicity. Such a situation exists with formaldehyde. The theoretical calculations coincided with experimental results in over 70 percent of the gas-particle combinations. The theory partially explains the potentiation of irritant gases by particulate material (often termed a synergistic effect), but subsequent research has shown that the problem is not as simple as had been assumed.

A synergistic effect was reported by Dalhamn and Reid [42] with ammonia and carbon particles. Ammonia is a highly soluble gas, so that synergism would have been predicted by LaBelle *et al.*[41] Rats were exposed for 60 days to 100 ppm ammonia alone, 7 mg/m^3 carbon alone, and to 119 ppm ammonia plus 3.5 mg/m^3 carbon. The carbon particles were 95 percent smaller than 3 μ and 65 percent smaller than 1 μ. In the group exposed to both ammonia and carbon, there was a high frequency of mucosal damage, and the ciliary activity seemed to be significantly impaired. The trachea of rats exposed to ammonia alone showed less severe damage, and the trachea was histologically normal in about 80 percent of the rats exposed to carbon alone.

Boren [43] exposed mice to carbon alone, to nitrogen dioxide, and to carbon which had previously been exposed to nitrogen dioxide. He stated that around 550 mg of nitrogen dioxide was adsorbed per gram of carbon. Samples of air taken from the chamber during the exposure of mice to carbon with adsorbed nitrogen dioxide indicated that there was about 25 ppm to 30 ppm free nitrogen dioxide. Although this is a high concentration of nitrogen dioxide, mice exposed to even higher concentrations (250 ppm) of nitrogen dioxide alone developed pulmonary edema, but neither single nor repetitive exposure produced parenchymal lung lesions. Control mice and mice exposed to carbon alone showed no anatomic abnormality of the lungs. Mice exposed to the carbon with adsorbed nitrogen dioxide developed focal destructive pulmonary lesions. The exposures in this group were 6 hours per day, 5 days a week, for 3 months.

The typical lesions of pneumonitis were observed by Gross *et al.*[44] in the lungs of hamsters, rats, and guinea pigs exposed to a "sufficiently large number of carbon particles with either adsorbed sulfur dioxide or nitrogen dioxide." Exposure was 8 hours per day, 5 days per week, for 4 weeks. The particle size of the carbon, and the meaning of the phrase "a sufficiently large number of carbon particles" were not given. There was no record of the amount of either gas adsorbed by the activated carbon particles. Histological sections were from animals killed about a month after the end of exposure, whereby it is concluded that the lesions were persistent. They were concentrated in the regions of the respiratory bronchioles and alveolar dusts and consisted of cellular wall thickening.

Pattle and Burgess [4] studied the effect of mixtures of sulfur dioxide and smoke on mice and guinea pigs. Their concentrations of sulfur dioxide were in the range of 2,700 mg/m^3 to 12,000 mg/m^3 (900 ppm to 4,000 ppm), and the smoke concentrations were in the range of 50 mg/m^3 to 135 mg/m^3. Their end point was the dosage required to produce death. With concentrations of this magnitude, the results obtained have little applicability to air pollution criteria. Although they found that the lethality of mixtures of sulfur dioxide and smoke was greater than the lethality of the sulfur dioxide alone, they considered the effect to be a simple additive one resulting from the action of smoke in blocking the bronchi and alveoli.

Salem and Cullumbine [5] studied the effect of kerosene smoke on the acute toxicity of sulfuric acid, sulfur dioxide, acrolein, and acetaldehyde in guinea pigs, mice, and rabbits. As in the work by Pattle and Burgess,[4] the concentrations were many magnitudes above those found in air pollution episodes. The administration of smoke prior to the exposure to the irritant substances did not alter the toxicity, although the effects of smoke on the toxicity of the irritants were highly variable when the two agents were given simultaneously. In guinea pigs, the toxicity of sulfuric acid was increased by the presence of smoke, the toxicity of acetaldehyde and sulfur dioxide was decreased, and the toxicity of acrolein was unchanged. In mice, the toxicity of sulfur dioxide was increased, while that of acetaldehyde and acrolein was decreased. In rabbits, the toxicity of acrolein and acetaldehyde was decreased by the smoke. The end point in all cases was the mean fatal dose.

A series of studies of the effect of hygroscopic particles on physiological response to irritants has been undertaken by Amdur,[22–27] using the pulmonary flow resistance technique. It was found initially that the response to sulfur dioxide was potentiated by particles of sodium chloride below 1 μ, but not by 2.5-μ particles, at concentrations of about 10 mg/m^3. With sulfur dioxide [24] and with formaldehyde [25] as irritants, decreasing the concentration of the aerosol, or the total dose of aerosol by shortening the exposure time,[22] decreased the degree of potentiation observed from the addition of sodium chloride. The hypothesis of LaBelle *et al.*[41] that irritant gases with high water solubility would be potentiated by particles did not explain adequately the data obtained. Sulfur dioxide, formaldehyde, acetic acid, and formic acid all have high water solubility, but it was found that the first two were potentiated by sodium chloride [23, 24] and that the latter two were not.[25, 27] Although both sulfur dioxide and formaldehyde were potentiated by sodium chloride, there are differences which suggest that the guiding mechanism in the case of sulfur dioxide may be chemical change and for formaldehyde may involve surface adsorption.[24]

Another paper [22] examines further the effect of various physical and chemical factors on the potentiation of sulfur dioxide by particulate material. The degree of potentiation observed could be correlated to some extent with the solubility of sulfur dioxide in the solutions of sodium chloride, potassium chloride, and ammonium thiocyanate.

Aerosols of soluble salts of ferrous iron, manganese, and vanadium, which had been shown by Johnstone and co-workers [45, 46] to be capable of catalyzing the conversion of sulfur dioxide to sulfuric acid when they were present as nuclei of fog droplets, showed a major potentiating action where present at concentrations of about 1 mg/m^3. On the other hand, dry manganese dioxide, activated or spectrographic carbon, iron oxide fume, open hearth dust, and triphenyl phosphate did not alter the response even when present in concentrations of 8 mg/m^3 to 10 mg/m^3.

It is clear from the data presented [22] that all particulate material does not potentiate the response to sulfur dioxide any more than one particulate (sodium chloride) has potentiated the response to all irritant gases tested. Both solubility of sulfur dioxide in a droplet and catalytic oxidation to sulfuric acid play a major role in the observed potentiation of sulfur dioxide by certain particulate matter.

D. CARCINOGENESIS

1. Carcinogens

The incidence of cancer, and the insidious nature of the onset of malignant cell activity, together make essential the utmost effort in determining whether factors associated with air pollution can lead to increased occurrence of lung cancer in susceptible individuals, or to increased susceptibility to cancer in the population at large. In various parts of the world, and especially in the United States, the relative mortality due to lung cancer has been increasing.[47] Furthermore, urban residents exhibit a greater liability to the development of lung cancer than do those living in rural areas,[48] and data from several investigations suggest that the epidemiological association between urban residence and lung cancer is of pathogenetic significance.[49-51] The association between lung cancer and cigarette smoking is too well documented to need further amplification here. There is, however, one feature common to both air pollution and cigarette smoking studies which should be emphasized. In no case, for the reasons explained in the introduction to this chapter, has a suspected carcinogen in fact been demonstrated experimentally to produce a lung tumor in man, although the epidemiological considerations to be developed in the next chapter may well show a significant association between the suspected material and cancer. Thus, substances such as the polynuclear aromatic hydrocarbons (of which benzo(a)pyrene, BaP, is the prime example) may or may not produce lung cancer in man. However, they do increase tumor incidence in laboratory animals and, in addition, have produced malignancies when in combination with specific particles or viral infection. The minimum possible risk should always be taken with a potentially toxic substance whose effect may appear after a long period of latency. The bulk of the ensuing discussion is therefore based on the hypothesis that carcinogens effective in animals may be significant in increasing human malignant tumor incidence, and the word "carcinogen" will be used without qualification.

A portion of the organic material present in the atmosphere as suspended particles (Table 1-2) may be carcinogenic, and carcinogenic materials have been identified in the atmosphere of virtually all large cities in which studies have been conducted. The incomplete combustion of organic matter is one of the major sources of such substances; the photochemical reaction products of aliphatic and aromatic constituents of gasoline in the presence of the atmospheric gases also possess some potency for generating tumors experimentally.

Chemical and physical studies of polluted urban air have been paralleled by carcinogenic investigations using skin painting, subcutaneous injection, and inhalation techniques. The carcinogenicity (as measured by these techniques) of extracts of materials collected from air as well as such pollutant sources as chimney soots, road dusts, and vehicular exhausts, has been established by many investigators. It should be noted first that, in many cases, use was made of mice (A-strain) particularly susceptible to tumor development; and second, only two of the studies, those involving ozonized gasoline, used inhalation as a route of administration. The observed response in the case of A-strain mice inhaling ozonized gasoline was the relatively rapid development of multiple adenomas in the lung which, although tumors, are not malignant.[52]

More recently, sequamous cell cancers of the lung were induced in C57 black mice following infection with influenza virus and continuous exposure to an aerosol of ozonized gasoline. The tumors produced were histologically identical with those observed most frequently in man.[53]

Measurable concentrations of inorganic substances, such as metal dusts and asbestos, demonstrated to be occupationally associated with increased liability to lung cancer development, are also emitted into the atmosphere.[54-60] Additional laboratory experimentation is needed to verify such carcinogenic potential relative to interacting effects and respective ambient atmospheric concentrations.

2. Polynuclear Aromatic Hydrocarbons as Carcinogens in Polluted Atmospheres

Polynuclear aromatic hydrocarbons are commonly regarded with extreme suspicion as possible carcinogens, and much of the experimental work performed has concerned these compounds. This section deals with the presence and stability of the hydrocarbons in the atmosphere, and with their elution from soots on which they may be adsorbed.

The concentration of benzo(a)pyrene (BaP) as a representative carcinogenic hydrocarbon in selected urban and nonurban areas within the United States is shown in Table 10–2;[61] in Table 10–3,[62] the benzo(a)-pyrene content is given as a fraction of the total aromatic hydrocarbon content of air pollutants of nine American cities.

Table 10–2.—BENZO(A)PYRENE CONCENTRATIONS IN SEVERAL URBAN AND NONURBAN AREAS.

State	μg BaP/1000 m^3 air	
	Urban	Nonurban
Alabama	24	0.076
Indiana	39	1.8
Maryland	14	0.70
Missouri	54	0.025
North Carolina	39	0.25
Oregon	8	0.01
Pennsylvania	61	1.9
South Carolina	24	1.1

Table 10–3.—BENZO(A)PYRENE AS A FRACTION OF THE TOTAL AROMATIC HYDROCARBON CONTENT OF SEVERAL URBAN ATMOSPHERES

City	BaP fraction of total aromatic hydrocarbon, μg/g	
	Lot 19	Lot 20
Atlanta	1,800	1,900
Birmingham	2,300	3,400
Cincinnati	2,800	5,200
Detroit	3,800	4,500
Los Angeles	660	260
Nashville	5,900	4,900
New Orleans	2,100	1,600
Philadelphia	2,500	?
San Francisco	680	290

The physical stability of aerosols in polluted atmospheres has already been described in general terms (Chapter 1). Chemically, benzo(a)pyrene seems to be relatively stable and, even in the presence of a strongly oxidizing atmosphere, such as that found in photochemical smog characteristic of Los Angeles, the rate of disappearance of benzo(a)pyrene is smaller than that of many other hydrocarbons. Table 10–4[63] shows the destruction under certain conditions of exposure of various polynuclear aromatic hydrocarbons present in air.

Polynuclear aromatic hydrocarbons may exist in the atmosphere adsorbed on carrier particles as well as in their free state. As was mentioned in Section C–2, in the respiratory system, particle size influences the rate and extent of elution of adsorbates either into the macrophages, or onto the respiratory epithelium. Polycyclic aromatic hydrocarbons cannot be readily eluted from soots of very small particle size; in fact, particles with an average diameter of less than 0.04 μ will remove these compounds from their immediate environment because of high surface adsorption. Particles above 0.04-μ average size range will generally release adsorbed aromatic polycyclic hydrocarbons in the presence of appropriate solvents, which include plasma and cytoplasmic proteins. As particle size increases, release becomes more rapid and greater in extent. The elution of polycyclic aromatic hydrocarbons from 0.5-μ soot particles after incubation with plasma for various time intervals is shown in Table 10–5.[64]

In studies on skin carcinogenesis, Falk and Steiner,[65] using commercial carbon blacks, related the biological activity of adsorbed carcinogens to the size of the soot particle and the presence of natural eluting substances at the site of deposition. They advanced the principles of natural and conditional carcinogens, of solvent elution, and of adsorption, to explain some clinical and epidemiological observations of human skin and lung cancers, and of the role of preceding pathological lesions in predisposing to pulmonary tumors.

Table 10-4.—EFFECT OF VARIOUS CONDITIONS OF EXPOSURE ON THE DESTRUCTION OF SOME POLYNUCLEAR AROMATIC HYDROCARBONS

Hydrocarbons	Percent destroyed						
	Pure unadsorbed					Adsorbed on soot	
	By air in dark		By air in light		By smog in light	By air in light	By smog
	Hours						
	24	48	24	48	1	48	1
Compound X	100		100		100	12	72
Anthanthrene	44	49	42	44		5	55
Phenanthrene	39	61	34	60			
Pyrene	24	43	20	42	83	1	58
Fluoranthene	17	20	16	24	59	4	59
3, 4 Benzpyrene	0	0	21	22	50	10	18
1, 2 Benzpyrene						7	51
1, 12 Benzperylene	0	0	0	0	27	0	67
Coronene	0	0	0	0	5		
Chrysene	0	0	11	0	15		

The interaction of carcinogens with other particulate agents has been studied toxicologically, utilizing laboratory animals. Experiments have shown that the addition of seemingly inert particulates to carcinogens results in the production of malignant neoplasms in the lung. Pylev[66] produced an appreciable incidence of lung cancers in rats by the intratracheal administration of 9, 10-dimethyl-1, 2 benzathracene (DMBA) incorporated with india ink in a 4-percent casein solution, whereas Gricute[67] was unsuccessful with the same material when it was suspended only in physiological saline. Saffiotti[68, 69] has produced a variety of malignant tumors in the lungs of hamsters by intratracheal instillation of saline suspensions of BaP ground together with hematite (Fe_2O_3) as a carrier dust in amounts equivalent to 3 mg of each chemical, once weekly for 15 injections. Not only was a high incidence of lung cancers produced but also these lung cancers mimicked all the various cell types seen in human cancers, i.e., squamous cell carcinoma, anaplastic carcinoma, adeno-carcinoma, and even tracheal cancers. Dose-response effects were suggested, as were indications that a single high dose could induce cancers in this system.[69] According to Saffiotti *et al.*[70] the increased action for the carcinogen is thought to be brought about by its adherence to the fine inert particulate which in turn carries it through the respiratory bronchioles and alveoli into the lung parenchyma. The carcinogens may then be eluted from the particulates and spread diffusely to reach the target tissue. They infer that there is also a reduction in the speed in which BaP is removed from the respiratory tract brought about by the hermatite dust. Shabad *et al.*[71] have reported that when india ink was included in the inoculum, BaP was eliminated more slowly from the lung. One must consider the possibility that the carrier material itself might be contributing some effect. Faulds and Stewart[72] reported increased incidence of carcinoma in the lungs of hematite miners. However, it is difficult to assess the exact role of iron in such a complicated exposure situation, since silica and other dusts are certain to be present. Bonser *et al.*[73] suggest that iron oxide may play a role in converting the fibrogenic effect of silica into a carcinogenic process. Haddow and Horning[74] have reported that the car-

Table 10-5.—PERCENTAGE RECOVERY OF POLYNUCLEAR AROMATIC HYDROCARBONS FROM 0.5μ SOOT PARTICLES AND FROM PLASMA AFTER INCUBATION WITH PLASMA FOR VARYING PERIODS.

Compound	Concentration, μg/mg Soot	Fraction	Incubation time, hours			
			1.5	16	96	192
			Percentage recovery			
Pyrene	8.4	Soot	9	34	9	6
		Plasma	81	61	50	61
		Total	90	95	59	67
Fluoranthrene	1.0	Soot	trace	present	0	present
		Plasma	100	present	present	present
		Total	100	—	—	—
Compound-X	2.8	Soot	0	trace	0	0
		Plasma	100	93	present	39
		Total	100	93	—	39
1,2-Benzpyrene	2.9	Soot	trace	17	0	0
		Plasma	52	41	21	21
		Total	52	58	21	21
3,4-Benzpyrene	1.7	Soot	18	41	6	18
		Plasma	82	59	23	18
		Total	100	100	29	36
1,12-Benzperylene	9.2	Soot	31	67	5	11
		Plasma	66	33	15	13
		Total	97	100	20	24
Anthanthrene	2.4	Soot	25	54	trace	13
		Plasma	54	33	17	13
		Total	79	87	17	20
Coronene	10.0	Soot	40	66	12	15
		Plasma	36	26	8	10
		Total	76	92	20	25

cinogenic action of an iron-dextran complex cannot be entirely explained by the dextran content alone. Consequently, iron cannot be excluded as a possible contributing factor or cofactor in cancer production. Epstein *et al.*[75] have recently applied methods employing the injection of crude benzene-soluble extracts of atmospheric particulate into suckling mice. The total dosages administered ranged from 5 to 55 mg and produced a high incidence of tumors in surviving mice as compared to controls. At 50 weeks post-inoculation, a variety of tumors was evident, the most significant being lymphomas, hepatomas, and multiple pulmonary adenomas. In mature mice injected subcutaneously with carcinogens, the tumors usually develop in the vicinity of the site of inoculation. However, in newborn mice, the tumors frequently develop at distal points such as the liver, lungs, or lymph nodes.

More recently, Kuschner[76] has demonstrated the interaction of a known carcinogen with the gaseous air pollutant, SO_2. Inhalation exposures of rats indicated that SO_2, alone, produced proliferative and metaplastic changes in the bronchial epithelium; benzo(a)pyrene, alone, failed to cause the development of tumors, but the inhalation of benzo(a)pyrene in the presence of SO_2 caused the development of bronchogenic squamous cell carcinoma.

3. Pathology of Carcinogenesis

Rather little experimental evidence is available on the pathology of carcinogenesis following exposure to air pollutants; much more experimentation has been carried out in relation to smoking and lung cancer. The results of the extensive work on the latter topic by Auerbach *et al.*[77] are reported briefly because of the possible similarities between carcinogenic mechanisms in cigarette smoking and air pollution. The authors describe a series of changes which are characterized by loss of cilia, increase in the number of cell rows, and the presence of atypical cells in the thickened epithelium. Their findings suggest a progression of changes from initial goblet cell hyperplasia to metaplasia, metaplasia with atypism, carcinoma *in situ*, and invasive cancer. Their data further support the epidemiologic observation of a dose response to cigarette smoke. Carnes[78] also observed a clear and pronounced association between the extent of epithelial change and exposure to pollutants, and emphasized the similarity of epithelial changes in man to "hyperplasia and other changes in the bronchial epithelium of mice." Indistinguishable histologic counterparts can be identified in both man and experimental animals.

The sequence of hyperplasia, metaplasia, metaplasia with atypical change, cancer *in situ*, and invasive cancer is likely to occur first at sites of impingement and particulate retention. In experiments with animals, particle deposition and retention occur most readily in the more distal segments of the tracheobronchial tree, a result which appeared at first to be incompatible with early clinical and pathologic observations that primary bronchial carcinomas originated chiefly in the main stem bronchi. Early clinical observations, however, included a majority of cases in which tumor size was great and point of origin was difficult to ascertain, and recent reports question the concept that most primary lung cancers are hilar or central in origin. For example, Table 10-6 shows the distribution of lung cancer by sites of origin given by Kotin.[79]

Table 10-6.—DISTRIBUTION OF LUNG CANCER BY SITE OF ORIGIN

Site of origin	Percentage of incidence
Main bronchus (including intermediate)	11
Lobar bronchus	29
Segmental bronchus	29
Segmental area (i.e., peripheral tumor)	31

E. SUMMARY

This chapter reviews toxicological studies of various types of particulate matter known to be present in ambient atmospheres. These studies are primarily concerned with the disciplinary areas of pathology, physiology, and carcinogenesis. In addition, the mechanisms of the toxicological action of particulate matter are discussed and considerations concerning mixtures of irritant gases and particulate matter are given proper emphasis. To date, studies utilizing laboratory animals have, in the main, been concerned with levels of particulate materials far in excess of ambient concentrations. Although direct extrapolations from the laboratory animal to man are impossible, animal experimentation is useful and necessary for rapidly defining the toxicological mechanisms of action and for pinpointing the primary biological systems affected by a specific particulate material alone or in the presence of an irritant gas. Findings obtained from animal experimentation can be tested under community conditions with human populations via epidemiological studies.

Particulate matter may exert a toxic effect via one or more of three mechanisms:

1. The particle may be intrinsically toxic due to its inherent chemical and/or physical characteristics;
2. The particle may interfere with one or more of the clearance mechanisms in the respiratory tract;
3. The particle may act as a carrier of an adsorbed toxic substance.

The last of these mechanisms can lead to a "potentiating" effect in which particles containing an adsorbed toxic substance increase the physiological response to the adsorbed substance to a level above that which one would expect if the substance were present in the absence of the particle. Conversely, prior exposure of an animal to particulate matter can sometimes afford a degree of protection to subsequent exposure to irritant gases. Particle size and dust load both contribute toward determining the toxicity of any specific chemical substances: reduction in particle size generally increases toxicity, while even an "inert" particle may elicit toxic responses when present in high enough concentrations. Finally, it has been clearly demonstrated that a number of substances (metal dusts, asbestos, polynuclear aromatic hydrocarbons, etc.) known to be carcinogenic in animals are present in polluted atmospheres. When linked with urban-rural differences in lung cancer frequency revealed from epidemiologic investigations, this evidence indicates that such substances in urban polluted atmospheres may be potential carcinogens for the exposed human population.

F. REFERENCES

1. Stokinger, H. E. and Coffin, D. L. "Biologic Effects of Air Pollutants." In: Air Pollution, Vol. I, A. C. Stern (ed.), Academic Press, New York, 1968, pp. 445–546.
2. Selikoff, I. J. and Hammond, C. "Tabular Data Analysis of the Evidence Suggesting General Community Asbestos Air Pollution." Preprint. (Presented at the Air Pollution Medical Research Conference, Denver, Colorado, July 1968.) Presented at the Air Pollution Medical Research Conference, Denver, Colorado, July 1968.
3. "Beryllium—Its Industrial Hygiene Aspects." H. E. Stokinger (ed.), Academic Press, New York, 1966.
4. Pattle, R. E. and Burgess, F. "Toxic Effects of Mixtures of Sulphur Dioxide and Smoke with Air." J. Pathol. Bacteriol., Vol. 73, pp. 411–419, 1957.
5. Salem, H. and Cullumbine, H. "Kerosene Smoke and Atmospheric Pollutants." Arch. Environ. Health, Vol. 2, pp. 641–647, 1961.
6. Wagner, W. D., Dobrogorski, O. J., and Stokinger, H. E. "Antagonistic Action of Oil Mists on Air Pollutants." Arch. Environ. Health, Vol. 2, pp. 523–534, 1961.
7. Amdur, M. O. and Devir, S. E. Unpublished data.
8. Schnurer, L. and Haythorn, S. R. "The Effects of Coal Smoke of Known Composition on the Lungs of Animals." Am. J. Pathol., Vol. 13, pp. 799–810, 1937.
9. Schnurer, L. "Effects of Inhalation of Smoke from Common Fuels." Am. J. Public Health, Vol. 27, pp. 1010–1022, 1937.
10. Pattle, R. E., Wedd, C. D., and Burgess, F. "The Acute Toxic Effects of Black Smoke." Brit. J. Ind. Med., Vol. 16, pp. 216–220, 1959.
11. Nau, C. A., Neal, J., and Stembridge, V. "A Study of the Physiological Effects of Carbon Black. I. Ingestion." Arch. Ind. Health, Vol. 17, pp. 21–28, 1958.
12. Nau, C. A., Neal, J., and Shembridge, V. "A Study of the Physiological Effects of Carbon Black. II. Skin Contact." Arch. Ind. Health, Vol. 18, pp. 511–520, 1958.
13. Nau, C. A., Neal, J., and Stembridge, V. "A Study of the Physiological Effects of Carbon Black. III. Adsorption and Elution Potentials." Arch. Environ. Health, Vol. 1, pp. 512–533, 1960.
14. Nau, C. A., Neal, J., Stembridge, V., and Cooley, R. N. "Physiological Effects of Carbon Black. IV. Inhalation." Arch. Environ. Health, Vol. 4, pp. 15–431, 1962.
15. Vintinner, F. J. and Baetjer, A. M. "Effect of Bituminous Coal Dust and Smoke on the Lungs. Animal Experiments." Arch. Ind. Hyg., Vol. 4, pp. 206–216, 1951.
16. Baetjer, A. M. and Vintinner, F. J. "The Effects of Silica and Feldspar Dusts on Susceptibility to Lobar Pneumonia. Animal Experiments." J. Ind. Hyg. Toxicol., Vol. 26, pp. 101–108, 1944.
17. Baetjer, A. M. "The Effect of Portland Cement on the Lungs with Special Reference to Susceptibility to Lobar Pneumonia." J. Ind. Hyg. Toxicol., Vol. 29, pp. 250–258, 1947.
18. Vintinner, F. J. "Effects of Aluminum Dust on Susceptibility to Lobar Pneumonia. Animal Experiments." Arch. Ind. Hyg., Vol. 4, pp. 217–220, 1951.
19. Amdur, M. O. "The Respiratory Responses of Guinea Pigs to Sulfuric Acid Mist." Arch. Ind. Health, Vol. 18, pp. 407–414, 1958.
20. Amdur, M. O. and Corn, M. "The Irritant Potency of Zinc Ammonium Sulfate of Different Particle Sizes." Am. Ind. Hyg. Assoc. J., Vol. 24, pp. 326–333, 1963.
21. Amdur, M. O., Silverman, L., and Drinker, P. "Inhalation of Sulfuric Acid Mist by Human Subjects." Arch. Ind. Hyg., Vol. 6, pp. 305–313, 1952.
22. Amudr, M. O. and Underhill, D. W. "The Effect of Various Aerosols on the Response of Guinea Pigs to Sulfur Dioxide." Arch. Environ. Health, Vol. 16, pp. 460–468, 1968.
23. Amdur, M. O. "The Influence of Aerosols upon the Respiratory Response of Guinea Pigs to Sulfur Dioxide." Am. Ind. Hyg. Quart., Vol. 18, pp. 149–155, 1957.
24. Amdur, M. O. "The Effect of Aerosols on the Response to Irritant Gases." In: Inhaled Particles and Vapours, Vol. I, C. N. Davies (ed.), Pergamon Press, London, pp. 281–292, 1961.

25. Amdur, M. O. "The Response of Guinea Pigs to Inhalation of Formaldehyde and Formic Acid Alone and with a Sodium Chloride Aerosol." Intern. J. Air Pollution, Vol. 3, pp. 201–220, 1960.
26. Amdur, M. O. "The Physiological Response of Guinea Pigs to Atmospheric Pollutants." Intern. J. Air Pollution, Vol. 1, pp 170–183, 1959.
27. Amdur, M. O. "The Respiratory Response of Guinea Pigs to the Inhalation of Acetic Vapor." Am. Ind. Hyg. Assoc. J., Vol. 22, pp. 1–5, 1961.
28. Nadel, J. A., Corn, M., Zwi, S., Flesch, J., and Graf, P. "Location and Mechanism of Airway Constriction after Inhalation of Histamine Aerosol and Inorganic Sulfate Aerosol." In: Inhaled Particles and Vapours, Vol. II, C. N. Davies (ed.), Pergamon Press, London, pp. 55–67, 1967.
29. Dautrebande, L. "Microaerosols." Academic Press, New York, 1962.
30. DuBois, A. D. and Dautrebande, L. "Acute Effects of Breathing Inert Dust Particles and of Carbachol Aerosol on the Mechanical Characteristics of the Lungs in Man. Changes in Response after Inhaling Sympathomimetic Aerosols." J. Clin. Invest., Vol. 37, pp. 1746–1755, 1958.
31. Amdur, M. O. "The Effect of High Flow-Resistance on the Response of Guinea Pigs to Irritants." Am. Ind. Hyg. Assoc. J., Vol. 25, pp. 564–568, 1964.
32. McDermott, M. "Acute Respiratory Effects of the Inhalation of Coal Dust Particles." J. Physiol., Vol. 162, p. 53, 1962.
33. Amdur, M. O. and Creasia, D. A. "The Irritant Potency of M-Terphenyl of Different Particle Sizes." Am. Ind. Hyg. Assoc. J., Vol. 27, pp. 349–352, 1966.
34. Tremer, H., Falk, H. L., and Kotin, P. "Effect of Air Pollutants on Ciliated Mucus-Secreting Epithelium." J. Natl. Cancer Inst., Vol. 23, pp. 979–997, 1959.
35. Stockinger, H. E. "Toxicologic Perspective in Planning Air Pollution Studies." Am. J. Public Health, Vol. 13, pp. 742–751, 1953.
36. Vorwald, A. J. "Effect of Inert Dusts in Air Pollution." In: U.S. Technical Conference on Air Pollution, McGraw-Hill, New York, 1952, pp. 485–488.
37. Schrenk, H. H. "Causes, Constituents and Physical Effects of Smog Involved in Specific Dramatic Effects." Arch. Ind. Hyg., Vol. 1, pp. 189–194, 1950.
38. Dautrebande, L. "Bases experimentales de la protection contre les gas de combat." J. Duculot (Belgium), 1939.
39. Dautrebande, L. and Capps, R. "Studies on Aerosols. IX. Enhancement of Irritating Effects of Various Substances in the Eye, Nose, and Throat by Particulate Matter and Liquid Aerosols in Connection with Pollution of the Atmosphere." Arch. Intern. Pharmacodyn., Vol. 82, 505–527, 1950.
40. Dautrebande, L., Shaver, J., and Capps, R. "Studies on Aerosols. XI. Influence of Particulate Matter on Eye Irritation Produced by Volatile Irritants and Importance of Particle Size in Connection with Atmospheric Pollution." Arch. Intern. Pharmacodyn., Vol. 85, pp. 17–48, 1951.
41. LaBelle, C. W., Long, J. E., and Christofano, E. E. "Synergistic Effects of Aerosols. Particulates as Carriers of Toxic Vapors." Arch. Ind. Health, Vol. 11, pp. 297–304, 1955.
42. Dalhamn, T. and Reid, L. "Ciliary Activity and Histologic Observations in the Trachea after Exposure to Ammonia and Carbon Particles." In: Inhaled Particles and Vapours, Vol. II, C. N. Davies (ed.), Pergamon, London, 1967, pp. 299–306.
43. Boren, H. C. "Carbon as a Carrier Mechanism for Irritant Gases." Arch. Environ. Health, Vol. 8, pp. 119–124, 1964.
44. Gross, P., Rinehart, W. E., and DeTreville, R. T. "The Pulmonary Reactions to Toxic Gases." Am. Ind. Hyg. Assoc. J., Vol. 27, pp. 315–321, 1967.
45. Johnstone, H. F. and Coughanowr, D. R. "Absorption of Sulfur Dioxide from Air Oxidation in Drops Containing Dissolved Catalysts." Ind. Eng. Chem., Vol. 50, pp. 1169–1172, 1958.
46. Johnstone, J. F. and Moll, A. J. "Formation of Sulfuric Acid in Fogs from Sulfur Dioxide in Air Containing Nuclei of Manganese and Iron Salts." Preprint. (Presented at the 136th American Chemical Society meeting, Atlantic City, New Jersey, 1959.)
47. Pascua, M. "Increased Mortality from Cancer of Respiratory System." Bull. World Health Organ., Vol. 12, pp. 687–704, 1955.
48. Kotin, P. and Falk, H. L. "The Role and Action of Environmental Agents in the Pathogenesis of Lung Cancer: I. Air Pollutants." Cancer, Vol. 12, pp. 147–163, 1959.
49. Kotin, P. "The Role of Atmospheric Pollution in the Pathogenesis of Pulmonary Cancer. A Review." Cancer Res., Vol. 16, pp. 375–393, 1956.
50. Cambell, J. A. "Cancer of the Skin and Increase in Incidence of Primary Tumors of Lung in Mice Exposed to Dust Obtained from Tarred Roads." Brit. J. Exptl. Pathol., Vol. 15, pp. 287–294, 1934.
51. Clemo, G. R., Miller, E. C., and Pybus, F. C. "The Carcinogenic Action of City Smoke." Brit. J. Cancer, Vol. 9, pp. 137–141, 1955.
52. Kotin, P. and Falk, H. L. "Experimental Induction of Pulmonary Tumors in Strain A Mice Following Their Exposure to an Atmosphere of Ozonized Gasoline." Cancer, Vol. 9, pp. 910–917, 1956.
53. Kotin, P. and Wiseley, D. V. "Production of Lung Cancer in Mice by Inhalation Exposure to Influenza Virus and Aerosols of Hydrocarbons." Progr. Exptl. Tumor Res., Vol. 3, pp. 186–215, 1963.

54. Hueper, W. C. "A Quest into Environmental Causes of Cancer of the Lung." U.S. Dept. of Health, Education, and Welfare, Public Health Service, Public Health Monograph 36, 1956.

55. Lynch, J. R. and Ayer, H. E. "Measurement of Asbestos Exposure." J. Occupat. Med., Vol. 10, pp. 21–24, 1968.

56. Balzer, J. L. "Industrial Hygiene for Industrial Workers." J. Occupat. Med., Vol. 10, pp. 25–31, 1968.

57. Tabershaw, I. R. "Asbestos as an Environmental Hazard." J. Occupat. Med., Vol. 10, pp. 32–37, 1968.

58. Cralley, L. J., Cooper, W. C., Lainhart, W. S., and Brown, M. C. "Research on Health Effects of Asbestos." J. Occupat. Med., Vol. 10, pp. 38–41, 1968.

59. Cralley, L. J., Keenan, R. G., Lynch, J. R., and Lainhart, W. S. "Source and Identification of Respirable Fibers." Am. Ind. Hyg. Assoc. J., Vol. 29, pp. 129–135, 1968.

60. Cralley, L. J., Keenan, R. G., Kuepel, R. E., and Lynch, J. R. "Characterization and Solubility of Metals Associated with Asbestos Fibers." Am. Ind. Hyg. Assoc. J., Vol. 29, p. 125, 1968.

61. Sawicki, E., Elbert, W. C., Hauser, T. R., Fox, F. T., and Stanley, T. W. "Benzo(a)pyrene Content of the Air of American Communities." Ind. Hyg. J., Vol. 21, pp. 443–451, 1960.

62. Hueber, W. C., Kotin, P., Tabor, E. C., Payne, W. W., Falk, H., and Sawicki, E. "Carcinogenic Bioassays of Air Pollutants." A.M.A. Arch. of Pathology, Vol. 74, pp. 89–115, 1962.

63. Falk, H. L., Markul, I., and Kotin, P. "Aromatic Hydrocarbons. IV. Their Fate Following Emission into the Atmosphere and Exposure to Washed Air and Synthetic Smog." A.M.A. Arch. Ind. Health, Vol. 13, pp. 13–17, 1956.

64. Falk, H. L., Miller, A., and Kotin, P. "Elution of 3, 4-Benzopyrene and Related Hydrocarbons from Soots by Plasma Proteins." Science, Vol. 127, pp. 474–475, 1958.

65. Falk, H. L. and Steiner, P. E. "The Identification of Aromatic Polycyclic Hydrocarbons in Carbon Blacks." Cancer Res., Vol. 12, pp. 30–39, 1952.

66. Pylev, L. N. "Experimental Production of Pulmonary Cancer in Rats by Intratracheal Introduction of 9, 10-dimethyl-1, 2-Benzanthracene." Biull. Eksp. Biol. Med., Vol. 52, pp. 99–102, 1961. (In Russian)

67. Greezuitay, L. A. "On the Mechanism of Blastomogenic Action of Urethane." Vopr. Onkol., Vol. 2, pp. 671–678, 1956. (In Russian)

68. Saffiotti, U., Cefis, F., Kolb, L. H., and Grote, M. J. "Intratracheal Injections of Particulate Carcinogens into Hamster Lungs." Proc. Am. Assoc. Cancer Res., Vol. 4, p. 59, 1963.

69. Saffiotti, U., Cefis, F., and Kolb, L. H. "Bronchogenic Carcinoma Induction by Particulate Carcinogens." Proc. Am. Assoc. Cancer Res., Vol. 5, p. 55, 1964.

70. Saffiotti, U., Cefis, F., Kolb, L. H., and Shubik, P. "Experimental Studies of the Conditions of Exposure to Carcinogens for Lung Cancer Induction." J. Air Pollution Control Assoc., Vol. 15, pp. 23–25, 1965.

71. Shabad, L. M., Pylev, L. N., and Kolesnichenko, T. S. "Importance of the Deposition of Carcinogens for Cancer Induction in Lung Tissue." J. Natl. Cancer Inst., Vol. 33, pp. 135–141, 1964.

72. Faulds, J. S. "Carcinoma of the Lung in Hematite Miners." J. Pathol. Bacteriol., Vol. 72, pp. 353–366, 1956.

73. Bonser, G. M., Faulds, J. S., and Stewart, M. J. "Occupational Cancer of the Urinary Bladder in Dyestuffs Operatives and of the Lung in Asbestos Textile Workers and Iron-Ore Miners." Am. J. Clin. Pathol., Vol. 25, pp. 126–134, 1955.

74. Haddow, A. and Horning, E. S. "On the Carcinogenicity of an Irondextran Complex." J. Natl. Cancer Inst., Vol. 24, pp. 109–147, 1960.

75. Epstein, S. S., Joshi, S., Andrea, J., Mantel, N., Sawicki, E., Stanley, T., and Tabor, E. C. "The Carcinogenicity of Organic Particulate Pollutants in Urban Air after Administration of Trace Quantities to Neonatal Mice." Nature, Vol. 212, pp. 1305–1307, 1966.

76. Kuschner, M. "The J. Burns Amberson Lecture: The Causes of Lung Cancer." Am. Rev. Respirat. Diseases, Vol. 98, pp. 573–590, 1968.

77. Auerbach, O., Stout, A. P., Hammond, E. C., and Barfinkel, L. "Smoking Habits and Age in Relation to Pulmonary Changes. Rupture of Alveolar Septums, Fibrosis, and Thickening of Walls of Small Arteries and Arterioles." New Engl. J. Med., Vol. 269, pp. 1045–1054, 1963.

78. Carnes, W. H. "The Respiratory Epithelium in Relation to Selected Diseases and Environmental Factors." (Report to the U.S. Dept. of Health, Education, and Welfare) April 1964.

79. Kotin, P. and Falk, H. L. "Carcinogenesis of the Lung: Environmental and Host Factors." In: The Lung. Williams and Wilkins Co., Baltimore, Maryland, pp. 203–225, 1968.

Chapter 11

EPIDEMIOLOGICAL APPRAISAL OF PARTICULATE MATTER

Table of Contents

Page
A. INTRODUCTION 148
B. APPLICATION OF EPIDEMIOLOGY TO AIR POLLUTION 148
  1. Indices 148
  2. Cautions 148
C. INDICES OF HUMAN RESPONSE: THE EPIDEMIOLOGIC STUDIES 150
  1. Acute Episodes 150
    a. Mortality 150
    b. Morbidity 154
  2. Chronic (Long-Term) Air Pollution 156
    a. Day-to-Day Variations in Mortality and Morbidity 156
    b. Geographical Variation in Mortality 156
      1. Studies Based on Available Data 156
      2. Special Studies Involving the Collection of New Data 158
    c. Geographic Variations in Morbidity—Special Studies 161
    d. Morbidity—Incapacity for Work 164
  3. Studies of Children 165
  4. Studies of Pulmonary Function 167
  5. Studies of Panels of Bronchitic Patients 168
D. SUMMARY 169
E. REFERENCES 176

List of Figures

Figure
11–1 Mortality Figures for the January 1956 and December 1957 Smog "Episodes" in London 151
11–2 Mortality and Air Pollution in Greater London during the Winter of 1958-1959 152
11–3 Death Rates and Air Pollution Levels in Dublin, Ireland, for 1938-1949 155
11–4 Average Annual Death Rate from all Causes 160
11–5 Average Annual Death Rate from Asthma, Bronchitis, or Emphysema Indicated on the Death Certificate 160
11–6 Average Annual Death Rate from Gastric Cancer 161
11–7 Age-Standardized Morbidity Rate per 1,000 for Three Diseases in Japan 162
11–8 Effect on Bronchitic Patients of High Pollution Levels (January 1954) 169

List of Tables

Table		Page
11–1	Pollution Levels in Salford (seasonal daily averages)	159
11–2	Average Annual Death Rates Per 1,000 Population From All Causes According to Economic and Particluate Levels, and Age: White Males 50-69 Years of Age, Buffalo and Environs, 1959-1961	159
11–3	Average Annual Death Rates Per 100,000 Population from Chronic Respiratory Disease According to Economic and Particulate Levels, and Age: White Males 50-69 Years, Buffalo and Environs, 1959-1961	160
11–4	Average Annual Death Rates Per 100,000 Population for Gastric Cancer According to Economic and Particulate Levels: White Males 50-69 Years of Age, Buffalo and Environs, 1959-1961	161
11–5	Response of Telephone Workers in the U.K. and the U.S.A. to Air Pollution	163
11–6	Summary Table of Epidemiological Studies	171

L

Chapter 11

EPIDEMIOLOGICAL APPRAISAL OF PARTICULATE MATTER

A. INTRODUCTION

Health effects produced by atmospheric particulate matter are discussed in this chapter in terms of epidemiologic studies. Because particulate matter tends to occur in the same kinds of polluted atmosphere as sulfur oxides, few epidemiologic studies have been able adequately to differentiate the effects of two pollutants. It follows, therefore, that the studies presented in this chapter are frequently identical with those described in the companion document, *Air Quality Criteria for Sulfur Oxides*.

Epidemiologic studies, as distinguished from toxicologic or experimental studies, analyze the effects of pollution from ambient exposure on groups of people living in a community. Such studies have the advantage of examining illness where it occurs naturally, rather than in a laboratory, but carry the disadvantage of not being able to control precisely all the factors of possible importance. Nevertheless, the preparation of air quality criteria must rest on epidemiologic studies because of the very severe limitations of toxicologic and industrial studies for this purpose. Other countries, notably the Netherlands and Sweden, have based their air quality criteria solely on epidemiologic studies.

In determining whether or not an association is causal, consideration must be given to several aspects of association which include strength, consistency, specificity, temporality, biological gradient, plausibility, coherence, and analogy.[1] A judgment of the value of an epidemiologic study requires an understanding of these aspects. Many types of epidemiologic evidence suggest that air pollution may exert considerable influence on health, as well as on the "satisfaction with life," of major segments of the world population.

Several health indices are described in Section B–1; certain precautions which should be observed in the application of epidemiologic methods to air quality criteria are suggested in Section B–2. The studies themselves are listed in Section C, according to the index employed.

B. APPLICATION OF EPIDEMIOLOGY TO AIR POLLUTION STUDIES

1. Indices

Various indices of health may be used for correlation with air pollution by the oxides of sulfur. Among the possible indices are:

1. mortality (greater than expected)
    - (i) deaths from all causes
    - (ii) deaths from specific causes
    - (iii) deaths among the different age and sex groups
2. morbidity
    - (i) incidence of disease—chronic bronchitis, pulmonary emphysema, diffuse interstitial pneumonitis, cancer of respiratory tract, disease of remote organ (e.g., gastrointestinal, ophthalmic, and cardiovascular systems)
    - (ii) prevalence of diseases—same examples as for "incidence"
    - (iii) prevalence of respiratory symptoms (e.g., changes in quality and/or quantity of sputum production)
    - (iv) exacerbation of diseases—rhinorrhea, asthma, tracheobronchitis, and chronic illness, and

    enhancement of infection: pneumonia, sinusitis, otitis, mastoiditis
    (v) changes in clinical conditions (e.g., bronchitic patients)
3. changes in various aspects of lung function
    (i) ventilatory function—decrease in peak flow rate, decrease in spirometric volumes, impairment of flow-volume relationship, and increased airway resistance
    (ii) blood/gas distribution—impairment of lung-gas distribution
    (iii) blood/gas exchange—impairment of pulmonary blood-gas exchange
    (iv) increased work of breathing

Definitions of the various disease states are to be found in the glossary; most of the pulmonary function methods have been mentioned in Chapters 9 and 10.

The manner of presentation of the state of epidemiological knowledge of effects of particulate matter in the ambient atmosphere when accompanied by the oxides of sulfur is outlined in the Table of Contents.

2. Cautions

In the first place, as discussed in Chapter 1, methods of measurement vary from country to country and place to place. Results from the high volume sampler and the results from smoke stain calibration are very dissimilar. Also, coh values cannot be compared with $\mu g/m^3$.

Secondly, particle size plays an important part in the appropriateness of the measuring technique as it does in determining the site and effectiveness of deposition in the respiratory tract. (See Chapter 9.) However, size distribution of particles has nearly always been ignored in field studies.

Thirdly, pollution and health indices are not always measured over the same time periods. It is to be hoped that the pollution levels cited bear some relation to those extant during the time when the chronic disease states were developing. Further, acute effects require frequent short-term pollution measurements to enhance detection, while long-term chronic processes may be adequately related to long-term sampling intervals. Air pollution measurements useful in studies of acute health effects are becoming available; a less satisfactory situation exists for the long-term effects studied.

In many instances the possible role of cigarette smoking has not been considered. It is expected that future epidemiologic studies involving adults will routinely collect data on smoking habits of the study group. Other factors are significantly related to respiratory disease. These include occupational and other past exposures; infections, past and present; and allergy and heredity.

Few or no epidemiologic studies have been possible where the pollution challenge has been *limited* to particulate matter, unaccompanied by *significant* amounts of other pollutant substances. Indeed, most of the available conclusions link particulate levels with those of concurrently measured sulfur dioxide; some studies attempt statistical separation of the culpability of one factor from the other in the effects cited.

In seeking the possible effects on populations resident in areas of differing air pollution, factors such as smoking, type and conditions of employment, ethnic group, and mobility in response to experienced irritation or disease have sometimes been considered. There has, however, been a minimum of attention paid to the indoor or domestic environments and their potential contribution. Measurement of such indoor exposures might be difficult, but omission of the information could well modify the appraisal of the importance of particulate pollution.

Toxicologic studies indicate a specific potential of some particulate materials to produce human responses. The levels used in toxicologic studies are far higher than those found in the communities in the epidemiologic studies under review. Thus the actual responsibility of "particulate matter" for the community responses is uncertain, and it is sometimes necessary to invoke additional concepts; for example, the idea of synergism with other known or unkown ambient pollutants, or the idea that particulate matter is but an index of availability of some other substance(s) which are fully responsible for the effects reported.

Over a short period of time, mortality fluctuates considerably, and only a systematic, long-term approach will allow a valid determination of the real role of air pollution. Cassell *et al.*[2] have reviewed the problem of detecting peaks in mortality and relating them to any single variable. The danger with episodic studies is that short-term fluctuation in the death rate, when picked to coincide with an air pollution incident, may appear to be causally related; in the long term, however, numerous other unassociated peaks are found in both the death rates and the air pollution levels.

Specific substances encountered as respirable particles, and associated with disease entities (beryllium, asbestos, arsenic, vanadium, lead, airborne pathogens, etc.) are not assessed in this report. They deserve consideration, however, as exemplars of the modes of response of which the human is capable, and which may delay or confound the recognition of the importance of "particulate matter" inhalation. The demonstration of the importance of aerodynamic behavior rather than of mass or particle dimension for both impingement and retention of asbestos is striking. Long-delayed effects from relatively brief community dispersion exposures to asbestos and beryllium are significant (see Chapter 10). Furthermore, penetration of "poorly cleared" particles of asbestos to remote body sites suggests complex body transport mechanisms, differing organ or tissue sensitivities, and the need for evaluation of as yet untested relationships between disease entities and respirable offenders. These models confer some sense of urgency to establishing the relationships of human disease and dysfunction to air pollution by "general particulate matter."

The concept of "susceptible population" demands consideration. Human responses to toxicants, and to community air pollution, show wide variations, which contribute in no small way to the difficulty in assessing in a general manner the effects of pollutants. Since air quality criteria must, unless otherwise specified, consider "all" population rather than just major segments of it, studies must consider especially the impact of air pollution on the "most sensitive" responders. Many factors seem to enhance susceptibility or sensitivity to air pollution. These include being at the extremes of age (i.e., infants and the very old); having pre-existing chronic respiratory disease (e.g., pulmonary emphysema or chronic bronchitis); having pre-existing cardiovascular disease (functional capacity not defined); regularly smoking cigarettes; or living in overcrowded or depressed socioeconomic strata. Some of these factors have been singled out for attention in the references to be cited, and the level of pollutant said to have an "effect" may take cognizance of such special sensitivity.

The effects discussed are related insofar as possible to specific pollution over specific time intervals; it must be emphasized that lower values by no means imply a "no effect" level of the pollutants.

C. INDICES OF HUMAN RESPONSE: THE EPIDEMIOLOGIC STUDIES

1. Acute Episodes

a. *Mortality*

In conurbations, such as London, New York, Chicago, and Detroit, it has been possible to observe deviations from the moving averages of deaths during various seasons, and to relate such deviations to the coincident period levels of air pollutants.[3]

In writing about the Meuse Valley episode in 1936, Firket stated that if there were a similar phenomenon in London, some 3,200 death might occur.[4,5] Unfortunately, he was quite accurate in his estimate since, in December 1952, the world's most disastrous smog incident occurred in London, causing about 4,000 excess deaths throughout the Greater London area. Marked increases were noted in both respiratory and cardiovascular deaths (and for almost every cause except traffic accidents; presumably the smog was too thick for people to drive). Since some of the diseases such as lung cancer and tuberculosis were obviously existent before the pollution episode, much of the effect of the fog was clearly to hasten the death of people who were already ill. Detailed investigations were made of 1,280 post-mortem reports of persons who had died before, during, or shortly after the episode. No fatalities were found which could not have been explained by previous respiratory or cardiovascular lesions. In this episode as in others, the elderly and persons with pre-existing pulmonary and cardiac disease were most susceptible.

A number of investigations have analyzed and compared the various London episodes. The report by Brasser *et al.*[6] appears to cover all the episodes and to present a detailed analysis of each of these episodes, pointing out the importance of the duration of the maximum values. More recently, Joosting[7] has examined the relationship between the duration of maximum values of sulfur dioxide and smoke during air pollution episodes, as well as the differential relationship between sulfur dioxide and smoke levels and the resulting mortality.

Gore and Shaddick[8] and Burgess and Shaddick[9] reviewed acute "fog" episodes which occurred in London in 1954, 1955, 1956 (January and Decmeber), and 1957 (December), in terms of excess mortality above a moving average, related to the mean of daily readings at seven stations for smoke and SO_2. Figure 11–1 shows mortality figures for the January 1956 and December 1957 episodes. Somewhat differing patterns of onset of mortality rise, of age of population suffering most heavily, of deaths related to bronchitis and to other respiratory diseases, and of total deaths, were noted in these acute episodes.

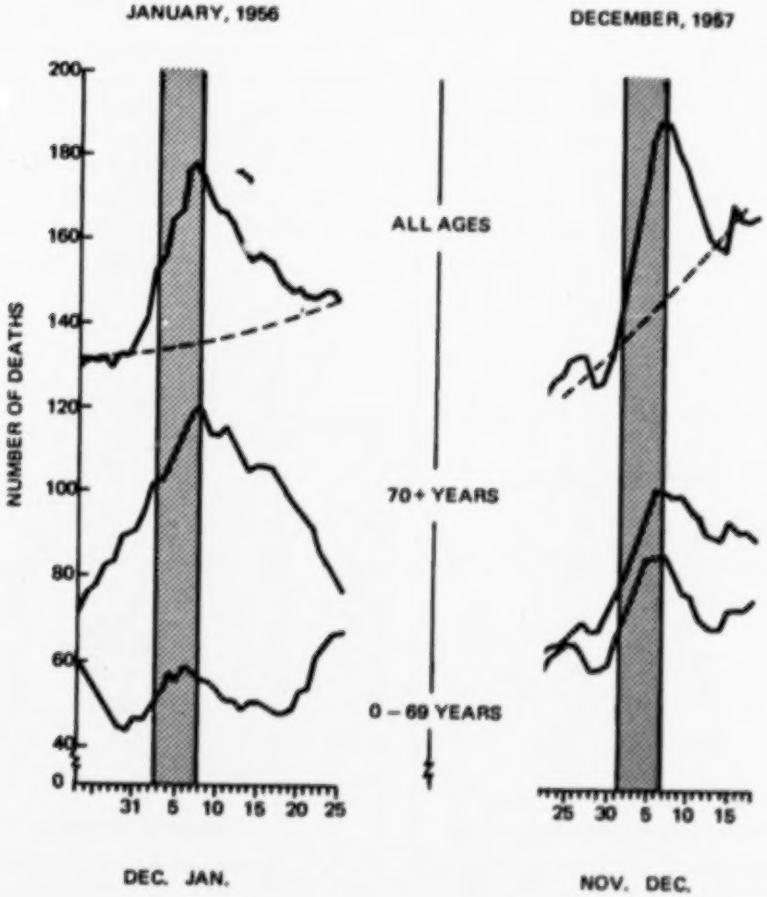

FIGURE 11–1. Mortality Figures for the January 1956 and December 1957 Smog "Episodes" in London.[9] (The figure shows the increase in numbers of deaths during smog "episodes" (shaded periods), especially in the older age group.)

Common to them, however, were elevations in the mean daily levels of SO_2 and smoke, measured at seven different stations, from two to four times the winter average levels; "effects" were estimated at 2,000 $\mu g/m^3$ black suspended matter together with 0.4 ppm (1,145 $\mu g/m^3$) SO_2 (representing all acidic gases). Deaths ascribed to bronchitis were materially affected, but deaths due to other causes also increased. Deaths appeared to begin to increase before the onset of the episodes; during the episodes, of course, they increased substantially. Scott[10] observed a similar relationship, for similar periods of "fog," with "effective pollutant" levels at seven different stations in London of 2,000 $\mu g/m^3$ for smoke and 0.4 ppm for SO_2. There was a sharp impact on the elderly and the greatest proportionate rise, for cause of death, in bronchitics.

Martin and Bradley[11] correlated daily mortality (all causes) and daily bronchitis mortality with mean daily black suspended matter (see Figure 11-2) measured at seven locations in London for the winter of 1958–1959, and also found a significant positive association between mean daily sufur dioxide levels and deaths (all causes). Bronchitis deaths showed a lower correlation with the pollution level, and the authors suggest the need for consideration of effects of air pollution on patients with cardiovascular disease. In addition, excess deaths have been related to increases (on the day preceding death) of mean daily black suspended matter by more than 200 $\mu g/m^3$, and rises in mean daily SO_2 of more than about 75 $\mu g/m^3$ (2.5 pphm). In a later paper,[12] data are shown to suggest an increase in mortality from all causes, and of respiratory and cardiac morbidity, associated with levels of smoke about 1,000 $\mu g/m^3$, and SO_2 concentrations of 715 $\mu g/m^3$ (25 pphm). This "effect" is properly related to abrupt rises in the concentration of smoke and/or SO_2, with perhaps a continuum of effects at lower levels. Since these measurements were obtained at a single point in Central London, it should be presumed that a relatively wide range of values around these levels actually contributed to the mortality statistics which were correlated. A reanalysis by Lawther[13] of these mortality studies

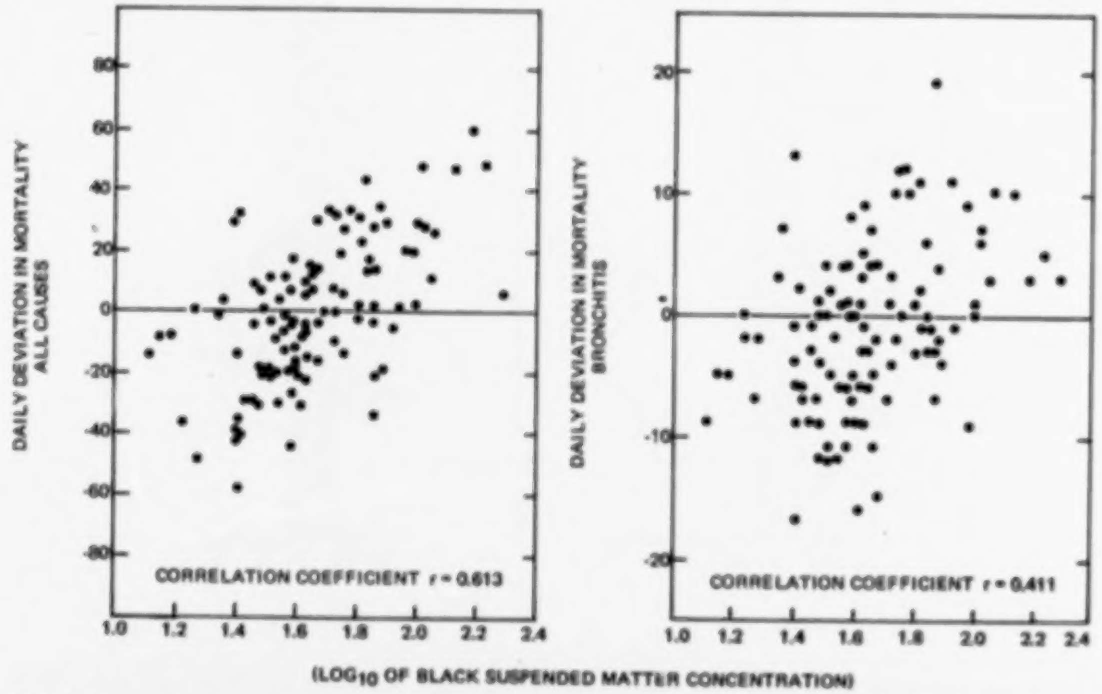

FIGURE 11-2. Mortality and Air Pollution in Greater London during the Winter of 1958-1959.[11] (The figure shows increased mortality due to "all causes" and to bronchitis during a period of high pollution.)

places the mortality "effect" at about 750 μg/m^3 for smoke and about 715 μg/m^3 (0.25 ppm) for SO_2. Joosting states that the maximum sulfur dioxide concentration above which significant correlation occurs with death and disease is 400 μg/m^3 to 500 μg/m^3 (0.15 ppm to 0.19 pppm)* when there is a high soot content.

The Dutch report on sulfur dioxide,[8] which discusses in detail seven air pollution episodes in London, states that in the December 1956 episode, 400 excess deaths, or 25 percent above expected, were observed in Greater London at maximum 24-hour levels of 1,200 μg/m^3 for smoke and 1,100 μg/m^3 (0.41 ppm)* for SO_2. The report also notes that in January 1959, 200 excess deaths were observed in Greater London, or 10 percent above expected mortality, at a level of 1,200 μg/m^3 for smoke and 800 μg/m^3 (0.30 ppm)* for SO_2. The episodes all took place during winter; cold weather seems to have been an important feature of London air pollution mortality.

In Martin's review [12] in 1964 of daily mortality in London during the winters of 1958–1959 and 1959–1960, he concluded: "From the data it would be difficult to fix any threshold value below which levels of air pollution might be regarded as safe." However, his review included data with smoke concentrations ranging upward from 500 μg/m^3 and accompanied by sulfur dioxide concentrations of about 400 μg/m^3 (0.14 ppm) and above.

As a result of smoke control regulations, the particle content of London air has steadily decreased since the 1950's, but the sulfur dioxide concentrations have not decreased proportionately. At the same measuring sites as in 1952, sulfur dioxide was actually slightly higher in the 1962 episode than in that of 1952, but smoke levels were considerably lower. Also, as Brasser *et al.*[8] have noted there was only one day of maximum pollution values in 1962 as contrasted with the 2 days of maximum pollution in December 1952. Although the smoke levels appear to be better related to the amount of excess mortality, other factors must be considered as possibly also reducing the number of deaths. Since 1952, a great deal of publicity has been given to the harmful effects of smog, and more susceptible individuals have been encouraged to use masks and filters and stay indoors. In addition, when episodes come close together, a large number of susceptible individuals might not accumulate, since some are killed off each time. An effect as large as that seen in the first incident would not, therefore, be expected.

The number of deaths in New York City was reviewed for excess mortality in relation to the air pollution episode of November 1953, by Greenburg *et al.*[14] Excess deaths were related to elevations of concentrations of sulfur dioxide and suspended particles. Average daily suspended particulate matter measured in Central Park was in excess of 5.0 coh units, while the SO_2 rose from the New York City average ranges of 430 μg/m^3 to 570 μg/m^3 (0.15 ppm to 0.20 ppm) to a maximum level of 2,460 μg/m^3 (0.86 ppm). For this episode, there was a "lag effect," and distribution of excess deaths among all age groups was noted. The number of deaths, although not showing the marked rise seen in some of the London episodes, was above average for comparable periods in other years during and immediately after the incident. For the November 15 to 24, 1953, period, the average number of deaths per day was 244, whereas during the three years preceding and following 1953, the average was 224 deaths per day for the same calendar period.

A later episode (1962) was studied,[15] But Greenburg *et al.* did not discern an excess mortality. However, McCarroll and Bradley,[16] reviewing episodes in New York City in November and December of 1962, January and February of 1963, and February and March of 1964, compared 24-hour average levels of various pollutants with New York City mortality figures, employing daily deviations from a 15-day moving average; the measurements were performed at a single station in lower Manhattan and fluctuations in the values at this station were known to correlate well with those at another station

---

*SO_2 is converted from ppm to μg/m^3 in the Dutch report by using the equivalency 2,700 μg/m^3=1 ppm; Scott apparently used 2,850 μg/m^3=1 ppm; this report uses 2,860 μg/m^3=1 ppm.

6.5 miles away. Excess deaths on December 1, 1962, followed a daily average SO_2 concentration of 2,060 $\mu g/m^3$ (0.72 ppm) and smoke shade in excess of 6 coh units, during a period of atmospheric inversion and low-ground-wind speed. The increased death rates were shared by the 45-to-64 age group, as well as by the age group over 65. In a later episode (January 7, 1963) associated with an SO_2 concentration of 1,715 $\mu g/m^3$ (0.6 ppm) and smoke shade at 6 coh units, there was a peak of death rate apparently superimposed upon an elevated death rate average due to the presence of influenza virus in the community. Two further episodes [16, 17] also suggest the relationship of excess deaths to abrupt rises in both SO_2 and suspended smoke at times of air stagnation.

An example of the inherent danger of relating mortality peaks to air pollution is shown by Leonard *et al.*[18] in the Dublin studies. During the war and post-war years of 1941 to 1947, peat was burned as the main fuel rather than coal, and air pollution (as measured by particle concentrations) was markedly decreased. Sulfur dioxide levels varied in a manner similar to those of suspended particulate matter. The winter peaks in death, however, were unaffected, and thus do not seem to be related to air pollution. When coal again became available in 1948, the air pollution levels rose with no apparent effect on the death rate (see Figure 11-3). Unfortunately, it is not possible to assess the effects of changing medical practices and the advent of antibiotics for use in treating respiratory diseases on these data.

In Detroit [19] a rise in infant mortality and deaths in cancer patients occurring over a 3-day period accompanied a rise in the 3-day mean suspended particulate matter for the same period above 200 $\mu g/m^3$ accompanied by an SO_2 maximum of 2,860 $\mu g/m^3$ (1.0 ppm) (September 1952). This is not believed to be related to the cold temperatures which have characterized the London episodes.

In Osaka, Japan, Watanabe [20] reported on excess deaths in a December 1962 smog episode. There were 60 excess deaths related to mean daily concentrations of suspended matter greater than 1,000 $\mu g/m^3$, with accompanying SO_2 greater than 285 $\mu g/m^3$ (0.1 ppm); the measurements were made at a single station in the central commercial area of the city.

When a marked increase in air pollution is associated with a sudden dramatic rise in the death rate or illness rate lasting for a few days and both return to normal shortly thereafter, a causal relationship is strongly suggested. Sudden changes in weather, however, which may have caused the air pollution incident, must be considered as another possible cause of the death rate increase. Over the years, a number of such acute episodes have been reported, and there seems little doubt that air pollution was the cause.

The British studies presented in this section suggest that excess mortality, a small rise in the daily death rate, is detectable in large populations if the concentrations of smoke and SO_2 rise abruptly to levels above 750 $\mu g/m^3$ and 715 $\mu g/m^3$ (about 0.25 ppm) respectively; the same effects are noted in American cities for SO_2 concentrations above 1,700 $\mu g/m^3$ (about 0.6 ppm) and a "smoke shade" of 6 coh units. The major targets are the aged population, patients with chronic obstructive pulmonary disease, and patients with cardiac disease. A more distinct rise in deaths is noted generally when particulate matter reaches about 1,200 $\mu g/m^3$ for one day and sulfur dioxide levels exceed 1,000 $\mu g/m^3$ (about 0.35 ppm). Daily concentrations of suspended particulates exceeding 2,000 $\mu g/m^3$ for one day in conjunction with levels of sulfur dioxide in excess of 1,500 $\mu g/m^3$ ($\sim$0.5 ppm) appear to be associated with an increase in the death rate of 20 percent or more over baseline levels.

b. *Morbidity*

The acute episodes have resulted in substantial increases in illness. Thus a survey [21] of emergency clinics at major New York City hospitals in November 1953 indicated a rise in visits for upper respiratory infections and cardiac diseases in both children and adults in all of the four hospitals studied. "Smoke shade" was close to 3 coh units, and sulfur dioxide concentrations had not yet exceeded 715 $\mu g/m^3$ (0.25 ppm) when hospital admissions clearly rose.[14]

Again, the number of emergency clinic

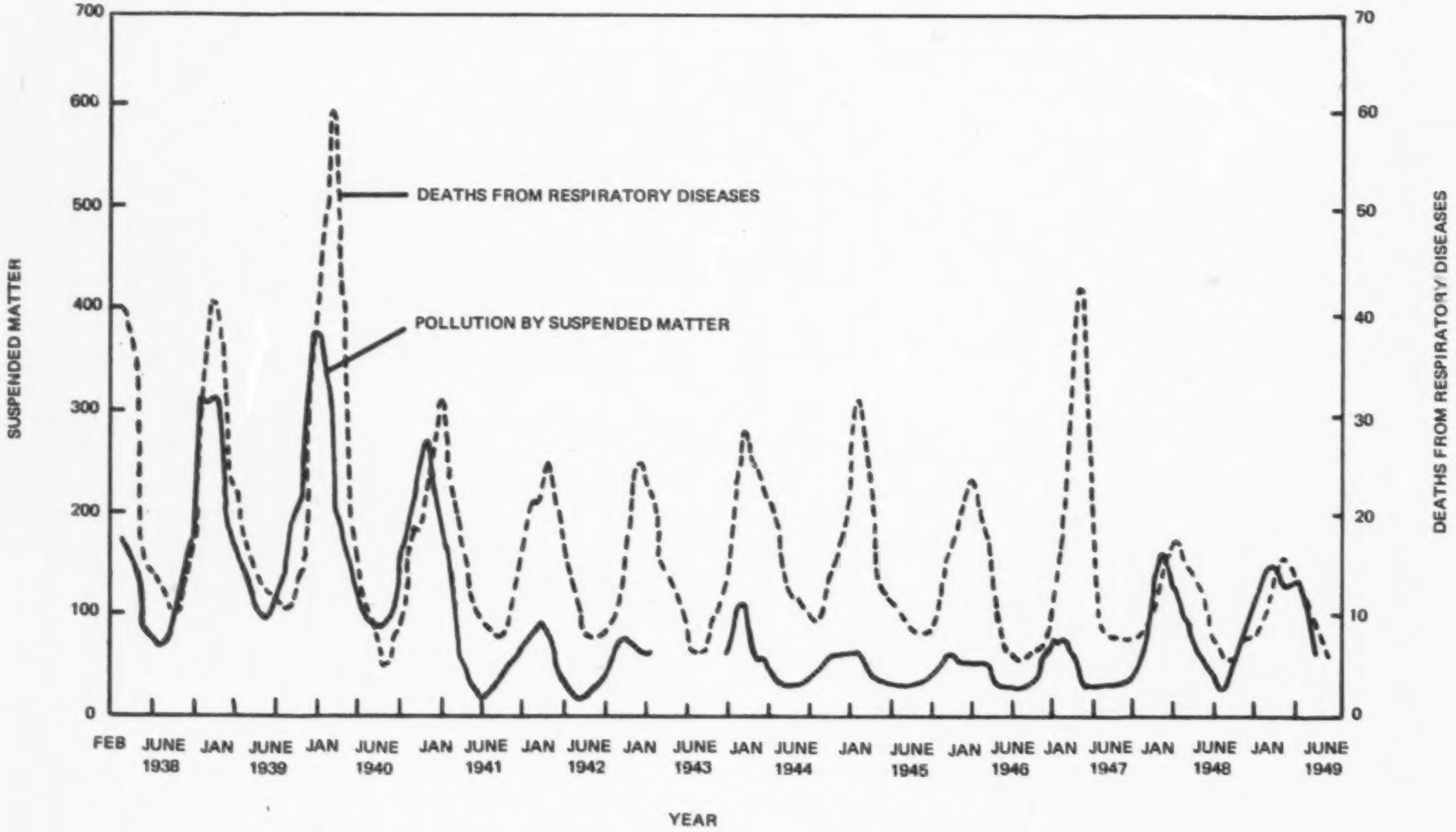

FIGURE 11–3. Death Rates and Air Pollution Levels in Dublin, Ireland for 1938-1949.[18]

visits for bronchitis and asthma at seven large New York City hospitals was examined during the Thanksgiving 1966 air pollution episode.[22] There was a rise in the number of such visits on the third day of the episode, among patients age 45 and over, at three of the seven hospitals investigated. Unfortunately, the Thanksgiving holiday greatly complicated evaluation of the emergency clinic visits over the holiday period.[17]

In the investigation of the London episode of December 1952, information on illness was collected from as many sources as possible including sickness claims, applications for hospital admission, pneumonia notifications and records of physicians. The analysis demonstrated a real and important increase in morbidity, though there was some indication that the increase in illness was not as large proportionately as the increase in deaths and the effects were not so sudden in producing a marked rise in the early days of the episode. In a number of other severe London episodes the increase in morbidity put a considerable strain on the health services.

These episodes reflect results which fall into Level IV of the World Health Organization's "guides to air quality."[23] These "guides," equivalent in usage to our term "criteria," cover sets of concentrations and exposure times at which specified types of effects are noted or at which no effect is noted. Level IV includes "concentrations and exposure times at and above which there is likely to be acute illness or death in susceptible groups of the population."

2. Chronic (Long-Term) Air Pollution

Kurland[24] has called to our attention that air pollution episodes represent, by definition, massive, overwhelming, and unusual exposure, and thus the most significant pathologic effects. There is an "iceberg" effect in that such data represent the obvious, while the greater share of the problem remains submerged. We are dealing with an essential dose-response situation, the upper limits of which are represented by these episodes.

a. Day-to-Day Variations in Mortality and Morbidity

In a systematic approach to analyzing respiratory and cardiac morbidity daily in London, Martin[12] examined deviations in morbidity from a 15-day moving average in London during the winters of 1958–1959 and 1959–1960. Both smoke and sulfur dioxide concentrations appear to be about equally related to morbidity rates, and a definite excess in morbidity seemed to exist as it did for mortality, though there was a somewhat greater degree of irregularity.

An approach similar to Martin's, but limited to observations on mortality, was used by McCarroll and Bradley[16] in New York City. Covering a 3-year period (1962–1964) they examined a number of peaks in New York City mortality associated with periods of high air pollution. There are examples given where sulfur dioxide and smoke shade appear to be related to mortality. The authors present other episodes, however, where the relationship to air pollution is not nearly as clear, although the death rate fluctuates to even higher peaks. Reference to this analysis has already been made in our discussion of the data on episodes in Section B.1.

McCarroll *et al.*[22] studied residents of a New York City housing project, using weekly questionnaires. Exact levels of air pollutants which could be used for establishment of air quality criteria were not given; however, the data indicate that sulfur dioxide rather than particulate matter was associated with eye irritation. Symptoms of cough were also shown to be related to air pollution but were not well differentiated between association with particles and sulfur dioxide. The particular time-series analysis used with these data is not well known, and the biases inherent in its use have not been fully determined.

b. Geographical Variations in Mortality

1. *Studies Based on Available Data.*—Mortality and morbidity statistics each have advantages as well as disadvantages. Records of illness should be more fruitful in defining subtle effects, since illness precedes death and since all illness does not result in death. Mortality statistics are collected in every country and are available for quick tabulation. Unfortunately, the quality of mortality statistics varies. One of the prob-

lems is that with the present system of tabulating mortality data, a single cause of death must be selected and coded, even though more than one cause may be involved in the death. The single cause of death designated (e.g., specific chronic respiratory disease) depends largely on the judgment of the attending physician and has little, if any, relation to epidemiologic use. While contributing causes of death appear on the death certificate, they are not reflected in summary tabulations. The coding of only the "underlying" cause of death minimizes the importance of such diseases as emphysema which often appear on the death certificate as contributory or associated causes of death.[25]

Almost all studies of the effects of long-term exposure on death rates compare the rate in one area with that in another. Mortality as well as morbidity studies are hampered by the possibility of differences other than air pollution existing between the areas, such as social class, occupation, age, and sex composition of the population, and cigarette smoking. Assuming that almost all deaths are recorded and tabulated, comparison of total mortality rates (i.e., deaths from all causes) obviates the bias of diagnostic selection, but does not lessen the chances of other associated factors having caused the difference.

Buck and Brown[26] reported in 1964 the relation of standardized mortality ratios for the 5-year period 1955-1959 to four variables: daily smoke and SO_2 concentrations for March 1962 (presumed representative of the study period), population density in 1961, and a social index of 1951. The studies involved populations in 214 areas of the United Kingdom (19 London boroughs, 49 county boroughs, 70 boroughs, 61 urban districts, and 15 rural districts).

Statistical studies indicate that bronchitis mortality had a significant positive association with both the smoke and the SO_2 concentrations encountered in these residential areas, and also with social index. The standardized mortality rates for lung cancer were not, in general, significantly associated with smoke or SO_2 concentrations in the residential areas. Examination of the tables given by Buck and Brown suggests that the excess of bronchitis mortality occurred for classes of area where the average daily smoke and SO_2 concentrations both exceeded 200 $\mu g/m^3$. Although smoking habits were reviewed and were apparently uniform from area to area, occupational and domestic indoor environmental exposures were not considered. The pollutant values selected for the correlations do not cover the same time period as the mortality figures.

A series of papers by Stocks *et al.*,[27–31] related atmospheric pollution in urban and rural localities with mortality due to lung cancer, bronchitis, and pneumonia. Standardized mortality ratios apparently refer to the period 1950 to 1953 for bronchitis and pneumonia and to the period 1950 to 1954 for lung cancer. Lung cancer mortality was found to be strongly correlated with smoke density in the atmosphere for 26 areas of northern England and Wales, for 45 districts of Lancaster and Yorkshire, and for 30 county boroughs. (Similar but weaker correlations were observed within Greater London.) Further, social differences in the populations concerned only partially explain this correlation. Bronchitis and pneumonia for males and bronchitis for females similarly showed strong correlations with smoke density in the atmosphere. Cancers of the stomach and intestine in the county boroughs were also related significantly to smoke concentrations, and other relationships are described for various areas of the country. It is, however, difficult to extract specific quantitative "effect" levels for smoke or the other pollutants studied. Papers[30, 31] describe the statistical elimination applied to develop significant correlations of spectographic analyses of 13 trace elements with mortality rates. Reanalysis of the data by Anderson[32] confirmed the importance of smoke levels, as well as social and population parameters, to mortality from lung cancer; vanadium is also identified as an important independent contributor to lung cancer, female cancer, and pneumonia mortalities.

In summary, the results of this analysis of long-term mortality indicate effects which would coincide with Level III of the World Health Organization's "guides to air quality."[23] Level III is defined as "concentrations and exposure times at and above which there is likely to be impairment of vital physiologic functions or changes that may lead to chronic diseases or shortening of life."

2. *Special Studies Involving the Collection of New Data.*—In 1964, also, Wicken and Buck [33] reported on a study of bronchitis and lung cancer mortality in six areas of Northeast England, one in Eston, another in Stockton and four in rural districts. The deaths covered the period 1952 to 1962. The survey of decedents with cause of death from bronchitis or lung cancer was matched against the survey of decedents with cause of death from nonrespiratory disease controlled for age and sex; the basis for the diagnostic classifications was not stated in the report. Personal interviews were carried out with next of kin. Personal interviews of a random sample of households were also conducted to obtain sex, age, smoking habits and occupation of the population at risk, the living population. The survey of decedents was carried out between January and October 1963; the survey of the living population was carried out between December 1963 and March 1964. Smoke and sulfur dioxide concentrations were measured in the Eston urban district. One year's aerometric data was obtained. The study was excellent in principle though, unfortunately, sulfur dioxide and particulate values were available only for the Eston urban district.

Eston, itself, as a sub-study, was subdivided into North Eston and South Eston. North Eston contains or lies near heavy industrial plants, whereas South Eston is a residential area. During the period May 1963 to April 1964, mean weekly observations of the sulfur dioxide and smoke concentrations were carried out in two sites in North Eston and one station in South Eston. Smoke values were 160 $\mu g/m^3$ and 80 $\mu g/m^3$ for North and South Eston respectively. The sulfur dioxide value in North Eston on the yearly average was 115 $\mu g/m^3$ (0.040 ppm) and for South Eston it was 74 $\mu g/m^3$ (0.026 ppm). The deaths studied occurred between 1952 and 1962. Adjustments were made for differences in age composition, smoking habits, and social class, and these were insufficient to explain the differences in lung cancer and bronchitis mortality rates between the two localities. Occupational exposure to pollution was then taken into account in the analysis. The conclusion was that there is an association between the degree of air pollution and the incidence of lung cancer and bronchitis mortality in the two areas of the Eston urban district. Though both sulfur dioxide and smoke values and concentrations are furnished in the report and the effects apparently cannot be separated, Brasser *et al.*[6] apparently have used the sulfur dioxide concentration as the more relevant measure of this study.

The community of Salford was classified into three pollution areas by Burn and Pemberton,[34] according to Table 11–1. Five sampling stations in the area were employed. Despite the closeness of the ranges of values, a high rate of bronchitis mortality, of lung cancer mortality, and of deaths from all causes, was observed in the high, compared to the lower pollution wards. It appears (see Section C–4) that there was also an increased rate of bronchitis morbidity in the highly polluted wards.

Winkelstein *et al.*[35–37] analyzed pollution effects in a group of studies made in the Buffalo, New York, area. In July of 1961, 21 air sampling stations were set up in and around the city of Buffalo, and daily values for suspended particulates, dustfall, and oxides of sulfur were taken until June of 1963. Suspended particulate levels were used as the index of air pollution. On the basis of the measurements, the study area was divided into four contiguous areas according to their levels of air pollution. Level 1 was less than 80 $\mu g/m^3$ (2-yr. geometric mean); level 2, 80 $\mu g/m^3$ to 100 $\mu g/m^3$; level 3, 100 $\mu g/m^3$ to 135 $\mu g/m^3$; and level 4, greater than 135 $\mu g/m^3$. Each area was also divided into five economic groupings. Mortality figures for the area were taken from the period 1959 to 1961, with the 1960 census providing the population basis.

Annual death rates per 100,000 population for (1) all causes of death; (2) chronic respiratory disease; (3) malignant neoplasms of the bronchus, trachea, and lung; and (4) gastric carcinoma were related to the four areas of air pollution intensity and

Table 11-1.—POLLUTION LEVELS IN SALFORD (SEASONAL DAILY AVERAGES)[34]

Pollution area classification	Smoke $\mu g/m^3$		SO_2 $\mu g/m^3$ (ppm in parentheses)		Deaths $\frac{\text{observed}}{\text{expected}} \times 100$		
	Winter	Summer	Winter	Summer	All causes	Bronchitis	Lung cancer
High	680	270	715 (0.25)	255 (0.09)	106	128	124
Intermediate	490	170	460 (0.16)	200 (0.07)	100	97	84
Low	450	170	340 (0.12)	145 (0.05)	90	52	79

Note: The data in the original report show SO_2 concentrations in parts per hundred million (pphm).

to the socioeconomic levels of the deceased.

Among white males between 50 and 69 years of age, the death rate from all causes was 20 per 1,000 in the area with the least suspended particulates (less than 80 $\mu g/m^3$). It was 20 percent higher in the next most polluted area (between 80 $\mu g/m^3$ and 100 $\mu g/m^3$) and twice as high in the most polluted area (more than 135 $\mu g/m^3$). See Table 11-2 and Figure 11-4.

Among white males between 50 and 69 years of age, the death rate from chronic respiratory disease was 42 per 100,000 population for those living in the area with the least suspended particulates, 40 percent higher for those living in the next most polluted area, and about three times as high for those living in the most polluted area. See Table 11-3 and Figure 11-5. Suspended particulate levels did not appear related to deaths from cancer of the bronchus, trachea, or lung. However, positive correlations were established with suspended particulate levels and total mortality, and mortality from chronic respiratory disease, regardless of the economic level of the deceased. Moreover, a positive correlation was established between suspended particulate levels and gastric cancer that appeared to be independent of economic status. It should be noted, however, that this study did not take into account the smoking habits of the deceased.

Among white males and females between 50 and 69 years of age, the death rate from gastric carcinoma was 27 per 100,000 population for those living in the area with the least suspended particulates, half again as high for those living in the next most polluted area, and one and one-half times as high for those living in the most polluted area. See Table 11-4 and Figure 11-6.

The Nashville Air Pollution Study by Zeidberg *et al.*,[38, 39] used sulfation and dustfall data from 123 sampling stations, and sulfur

Table 11-2.—AVERAGE ANNUAL DEATH RATES PER 1,000 POPULATION FROM ALL CAUSES ACCORDING TO ECONOMIC AND PARTICULATE LEVELS, AND AGE: WHITE MALES, 50-69 YEARS OF AGE, BUFFALO AND ENVIRONS, 1959-1961.

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	36	41	52	43
2	24	27	30	36	29
3	—	24	26	33	25
4	20	22	27	—	22
5 (high)	17	21	20	—	19
Total	20	24	31	40	26

dioxide concentrations and soiling indices from 36 stations. All codable deaths registered between 1949 and 1960 (32,067) were then distributed among census tracts rated according to high, moderate, and low pollution levels, and upper, middle, and lower economic classes, and then further coded by age, sex, race, and underlying cause of death. Standardized mortality ratios (for total respiratory disease, and for pneumonia, influenza, bronchitis, emphysema, tuberculosis, and lung and bronchial cancer) were then

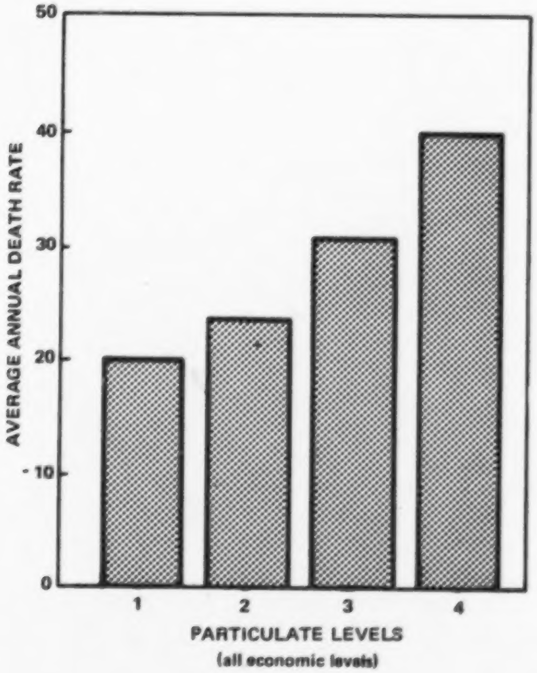

FIGURE 11–4. Average Annual Death Rate from All Causes. (The graph shows the death rate per 1,000 population for white males between 50 and 69 years of age for four levels of particulate matter, Buffalo and environs, 1959–1961.)

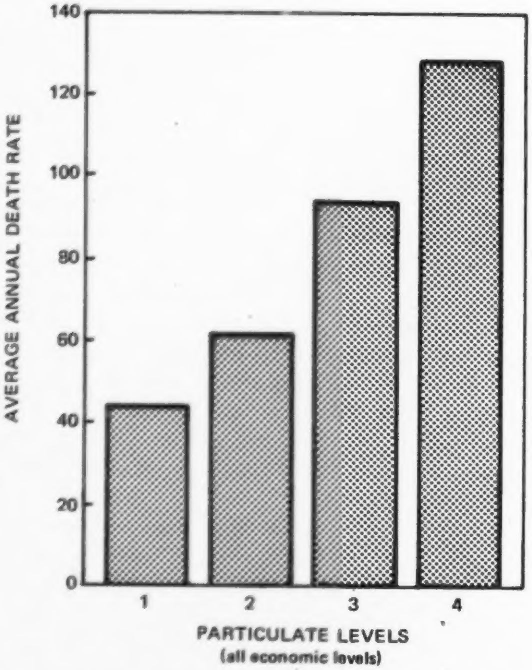

FIGURE 11–5. Average Annual Death Rate from Asthma, Bronchitis, or Emphysema Indicated on the Death Certificate. (The graph shows the death rate per 100,000 population for white males between 50 and 69 years of age for four levels of particulate matter, Buffalo and environs, 1959–1961.)

Table 11–3.—AVERAGE ANNUAL DEATH RATES PER 100,000 POPULATION FROM CHRONIC RESPIRATORY DISEASE ACCORDING TO ECONOMIC AND PARTICULATE LEVELS, AND AGE: WHITE MALES, 50–69 YEARS OF AGE, BUFFALO AND ENVIRONS, 1959–1961.

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	0[a]	126	188	133
2	64	75	96	105	84
3	—	65	51	103[a]	64
4	35	47	114	—	52
5 (high)	42	63	0[a]	—	50
Total	44	62	94	129	72

[a] Rate based on less than five deaths.

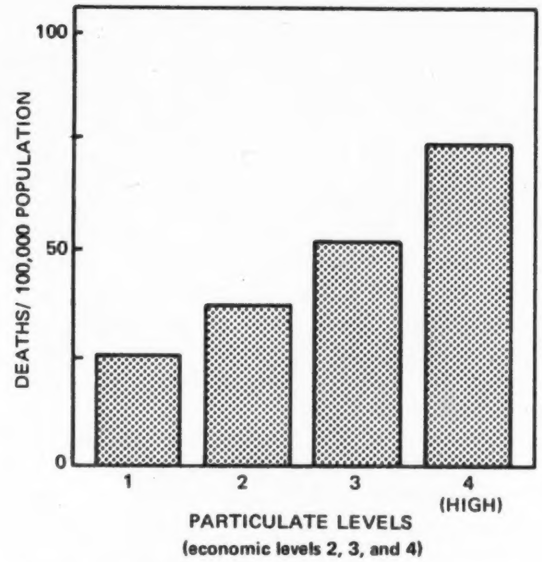

FIGURE 11–6. Average Annual Death Rate from Gastric Cancer. (The graph shows the death rate per 100,000 population for white males between 50 and 69 years of age for four levels of particulate matter, Buffalo and environs, 1959–1961.)

related to the pollution indices obtained during 1959. The statistically significant mortality increases were those for all respiratory diseases related to sulfation and soiling; lung and bronchial cancer mortality, and bronchitis and emphysema mortality were not clearly related. "High" pollution in these studies referred to soiling at more than 1.1 coh units/1,000 linear feet and SO_2 concentrations of more than about 30 $\mu g/m^3$ (.01 ppm). A later paper [40] derived from the same study period, analyzed infant and fetal death rates between 1955 and 1960. For white infant mortality, significant regressions were obtained for sulfation; dustfall (alone, or as an interaction variable) was the most frequently related variable. In the study, account was not taken of smoking habits of the deceased; also, the "middle class" group covered a relatively large segment of the decedents.

It is intresting to note that the association between suspended particulate levels and gastric cancer in the Buffalo study which appeared to be independent of economic status was also observed in the Nashville study.[38]

c. Geographic Variations in Morbidity—Special Studies

It has been postulated that the study of records of illness rather than mortality should be more fruitful in defining subtle effects, since morbidity is an earlier and more sensitive index of deviation from normal health. A much larger insult must presumably be given to the body to cause death than to cause illness. Routinely collected morbidity data are, however, not generally available. Data may occasionally be obtained from group insurance plans, hospital admission records, or existing school records. Since such data are usually not collected in a uniform, precise manner, most morbidity studies require expensive and time-consuming field surveys with questionnaires or actual medical examinations of the subjects.

Morbidity studies of adults involving long-term exposures are frequently not as useful

Table 11–4.—AVERAGE ANNUAL DEATH RATES PER 100,000 POPULATION FOR GASTRIC CANCER ACCORDING TO ECONOMIC AND PARTICULATE LEVELS: WHITE MALES, 50–69 YEARS OF AGE, BUFFALO AND ENVIRONS, 1959–1961.

Economic Level	Particulate Level				
	1 (Low)	2	3	4 (High)	Total
1 (low)	—	0(0)	63(10)	136(8)	77(18)
2	45(5)	41(12)	48(10)	84(8)	50(35)
3	—	39(9)	51(6)	51(2)	44(17)
4	15(3)	38(9)	63(5)	—	33(17)
5 (high)	26(5)	16(3)	0(0)	—	20(8)
Total	26(13)	34(33)	53(31)	—	42(95)

Figures in parentheses indicate numbers of deaths as desired due to the presence of complicating factors such as occupation and smoking. Accordingly, Anderson[30] has recommended that children and housewives be used to determine the health effects of air pollution.

A study was conducted by Petrilli *et al.*[41] in Genoa, Italy, which followed Anderson's recommendation. The subjects were women over 65 years of age, nonsmokers who had lived for a long period in the same area and who had no industrial work experience. Economic and social conditions in the areas of residence were thus considered as well as the levels of pollution in the areas of their residence. Pollutant measurements were carried out between 1954 and 1964 in 19 different areas, and morbidity indices were calculated for 1961 and 1962 for this population, which received free medical care from the municipality and therefore was under continuous medical observation. There was a significant correlation between the frequency of bronchitis with mean annual sulfur dioxide levels ($r=0.98$) and a nonsignificant correlation for mean annual suspended matter ($r=0.82$) and mean annual dustfall ($r=0.66$).

Toyama,[42] in a comprehensive study of air pollution and its health effects in Japan, charts the age-standardized morbidity rates (per thousand) secured by interview survey in 1961, and describes a gradient of respiratory disease morbidity from the highly industrialized (and presumably polluted) areas to the rural areas of Japan. Further, the pulmonary disease morbidity ratio was higher in the industrialized, polluted areas than were the ratios for other disease groupings. The gradient was not noted for cardiovascular diseases nor for gastrointestinal diseases. Unfortunately, specific pollutant concentrations are not clearly indicated to accompany these data on morbidity. The age-standardized morbidity rates per thousand for several cities in Japan are shown in Figure 11–7.

Holland *et al.*[43] studied the prevalence of chronic respiratory disease symptoms and performance of pulmonary function tests in a comparative study of outdoor telephone workmen in London, in rural England, and on the east and west coasts of the United States. Types of occupational exposure, use of cigarettes, and socioeconomic matching were considered. The annual mean concentration of suspended particulate matter in the British exposures was approximately 200 $\mu g/m^3$, and approximately 120 $\mu g/m^3$ in the American case. For sulfur dioxide, the mean concentrations were: in London 300 $\mu g/m^3$ ($\sim$0.1 ppm), in rural areas of England 60 $\mu g/m^3$ ($\sim$0.02 ppm), and in the United States between 30 $\mu g/m^3$ and 120 $\mu g/m^3$ ($\sim$0.01 ppm and $\sim$0.04 ppm). Persistent

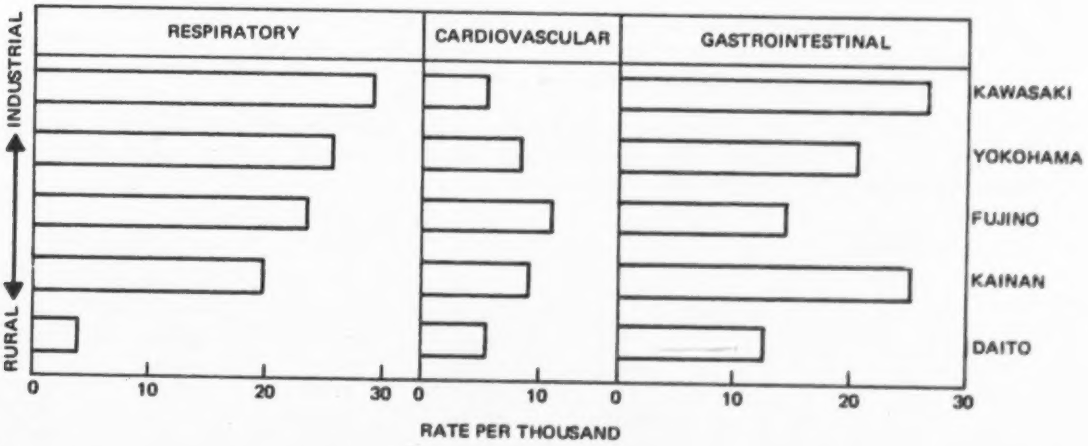

FIGURE 11–7. Age-Standardized Morbidity Rate per 1,000 for Three Types of Diseases in Japan.[42] (The mortality rate for respiratory diseases shows a clear correlation with pollution levels. There is no such clear correlation for cardiovascular or gastrointestinal disorders.)

cough, phlegm, and chest illness episodes and increased sputum volume were all significantly more frequent in men (between the ages of 50 and 59) in the London area than in rural England; and pulmonary function was poorer in relation to the levels of smoke and sulfur dioxide. It is possible, therefore, that an increase in smoke concentration from 120 μg/m³ to 200 μg/m³ (with an equivalent increase in sulfur dioxide level) increases the risk to older workers of deteriorated pulmonary function and increased symptomatology of chronic respiratory disease. Table 11-5 summarizes some of the results obtained.

Holland and Reid[44] reviewed respiratory symptoms, sputum production, and lung function levels in post office employees both in central London and in peripheral towns. Over the age of 50, the London men had more frequent and more severe respiratory symptoms, produced more sputum, and had significantly lower lung function tests. Socioeconomic factors were presumed the same, the occupational exposures were homogenous, and corrections were applied for smoking. There were some physique differences in the rural areas and allowances were made for these in the statistical evaluation. Unfortunately, no quantitative air quality determinations accompanied these results. The authors nevertheless concluded that the most likely cause of the difference in respiratory morbidity between the men working in Central London and those in the three rural areas was related to the differences in the local air pollution.

In a group of Canadian veterans studied by Bates *et al.*,[45] a relationship between air pollution and both bronchitis and pulmonary function measurements has been reported. There are, unfortunately, inadequate data on the levels of smoke, sulfur dioxide, and other pollutants, in the four Canadian cities compared, to derive specific relationships between the levels of these pollutants and the

Table 11-5.—RESPONSE OF TELEPHONE WORKERS IN THE U.K. AND U.S.A. TO AIR POLLUTION.[a]

	United Kingdom				United States			
	London		Rural areas		Baltimore Washington Westchester County		San Francisco Los Angeles	
	Age 40-49	Age 50-59	Age 40-49	Age 50-59	Age 40-49	Age 50-59	Age 40-49	Age 50-59
Number of men examined	113	137	267	159	396	229	361	119
Persistent cough and phlegm (percent)	25.7	38.7	24.0	18.9	22.2	25.8	21.6	24.4
Persistent cough and phlegm and chest illness episode (percent)	10.6	10.9	7.5	5.0	6.8	7.0	4.0	7.6
$FEV_{1.0}$ (liters) mean values:								
Non smokers	2.8	2.6	3.0	2.8	3.5	3.1	3.7	3.3
Cigarette smokers:								
1-14 per day	2.6	2.3	2.8	2.6	3.4	3.0	....	....
15-24 per day	2.6	2.2	2.8	2.5	3.2	2.8	3.4	2.8
25 or more per day	2.5	2.1	2.7	2.5	3.2	2.9	....	....
Sputum volume 2 cc. or more (percent)	28.9	42.9	22.1	23.5	7.1	10.0	8.6	14.3
Suspended particulate matter, 24 hr (μg/m³):								
Mean	220		200		120		120	
Maximum	4000		3000		500		340	
Sulfur dioxide, 24 hr (μg/m³), ppm in parentheses:								
Mean	290 (0.1)		60 (0.02)		110 (0.04)		30 (0.01)	
Maximum	3700 (1.3)		740 (0.26)		715 (0.25)		170 (0.06)	

prevalence or exacerbation of disease or the deterioration of pulmonary function. However, there is an association in the "dirty" cities (Montreal and Toronto) versus the "clean" cities (Halifax and Winnipeg) of increased prevalence and severity of bronchitis, and poorer pulmonary function performance.

Anderson and Ferris [46, 47] completed a comparison of respiratory disease incidence and air pollution in three residential areas of Berlin, New Hampshire, by prevalence survey. Subjects who were nonsmokers and those who were moderate smokers were specifically evaluated. The different levels of air pollution at one or more stations in each area were recorded in relation to monthly dustfall and to sulfur dioxide concentration. Prevalence of respiratory disease and pulmonary function determinations ($FEV_{1.0}$ and peak expiratory flow rate) were made. The mobility of the population, variations of concentrations of pollutants within a given area, possible effects from the area of occupation, and differences in the in-home environment, were considered as contributors to the lack of consistent effect on the studied parameters of air pollution. In a later study [48] these authors compared this "polluted" town in New Hampshire with a control community in British Columbia and again found an obscuring of the effects of air pollution on respiratory symptoms and pulmonary function performance, suggesting perhaps the overwhelming effect of cigarette smoking in symptom production. It should be noted, however, that the pollution levels and the ranges were possibly too low or too small to show a relationship.

The Nashville air pollution study [49] reviewed total morbidity in relation to air pollution. A wide matrix of sampling stations was used to group the residence of individuals into areas of high, moderate, and low pollution according to geometric mean annual measurements, and the morbidity data were secured by home interview. Significant "direct correlations" of *total morbidity* with levels of pollution (measured by soiling index and 24-hour sulfur dioxide levels) were observed for individuals over 55 in the middle socioeconomic class. Cardiovascular diseases were directly correlated with the aerometric parameters. There was no statistical evidence of increased respiratory disease morbidity or morbidity of other organs and systems. The same qualifications noted for the mortality data in this study apply to these data on morbidity.

d. *Morbidity—Incapacity for Work*

Dohan [50] reviewed the incidence of respriatory disease producing absence from work in a population of women employed in several branches of an electronics company in the eastern part of the United States. Pay scales were roughly equivalent and presumptions were made, therefore, that the socioeconomic status and smoking patterns were uniform. Air measurements were made from areas near the town of employment (for suspended particulate sulfates, nitrates, copper, zinc, vanadium, nickel, and acetone-soluble organic matter, as well as total suspended particulate matter). Absences for a respiratory illness in excess of 7 days were calculable from company-employment and health-insurance records. There was a significant correlation of respiratory disease absence frequency with the concentration of suspended particulate sulfates, with 24-hour values measured biweekly; although the level of total suspended particulate matter ranged from 100 $\mu g/m^3$ to 190 $\mu g/m^3$ in the various cities, a correlation was not demonstrated. The implication of the significantly correlated datum "suspended particulate sulfate" is not clear, as it might, for example, be an index of the community fuel consumption or an index of sulfur dioxide irritation.

Burn and Pemberton [51] reviewed the increased number of certificates of incapacity issued to workers in Salford in relation to smog episodes. When mean daily smoke pollution, based on data from five sampling stations, exceeded 1,000 $\mu g/m^3$ for 2 consecutive days, the number of "bronchitis certificates" issued exceeded the expected number by a factor of two, on four or five occasions in 1958.

During 1961–1962 a study of the incidence of incapacity for work was conducted by the British Ministry of Pensions and National Insurance.[51] The population covered was representative of the working population of England and Wales. Rates of sickness absence for bronchitis, influenza, arthritis and rheumatism were related to indices of pollution. There was a significant correlation between bronchitis incapacity in middle-aged men (35–54) and the average seasonal (October through March) levels of smoke and sulfur dioxide in high-density residential districts, based on 24-hour measurements. For Greater London, there was a significant correlation between bronchitis incapacity and *both* smoke and sulfur dioxide for all age groups taken together, and for men aged 35 to 54 and 55 to 59. It is interesting that there was also more incapacity from arthritis and rheumatism in areas having heavy smoke pollution. Influenza incapacity was greater in those areas with higher pollution levels over Great Britain as a whole but not within the Greater London conurbation, nor was there in this latter area any association between pollution and psychosis or psychoneurosis. The lowest bronchitis inception rates related to smoke levels between 100 $\mu g/m^3$ and 200 $\mu g/m^3$ and to sulfur dioxide concentrations between 150 $\mu g/m^3$ and 250 $\mu g/m^3$ (0.053 ppm and 0.081 ppm). The highest values related to particulate concentrations of 400 $\mu g/m^3$ and 400 $\mu g/m^3$ (0.14 ppm) sulfur dioxide.

Verma *et al.*,[52] presented information on illness absences in relation to air pollution. Illness data for the employees (males and females, ages 16 through 64) of a metropolitan New York insurance company were obtained through the records of the personnel department. They included medical history, X-ray information, and laboratory results obtained by the medical department of the company, and were classified by absences due to respiratory illness and to nonrespiratory illness. Mean daily concentrations of air pollution and meteorologic data were secured from the monitoring system of metropolitan New York: smoke shade, sulfur dioxide, and carbon monoxide content were reported. The data for the 2 years 1965 and 1966 were examined statistically and several conclusions were reached. There was a strong time dependence, and yearly cyclical behavior; when this factor was removed there remained *no* strong positive relationship between respiratory absence and the pollution variables studied. Respiratory illness absence rates were at their highest level when sulfur dioxide and smoke shade levels were both high on cool days; and a lag effect for respiratory absences was *not* noted.

3. Studies of Children

Comparisons of the prevalence of respiratory disease in areas of varying pollution levels have been made to delineate the role of air pollution and specific pollutants. A problem common to all the studies is the difficulty in guaranteeing that the areas are similar (except for air pollution) in all factors that might affect the prevalence of disease.

Because studies on adults tend to be complicated by smoking habits, changes of occupations, and changes in address over a period of years, several studies have been directed at effects of air pollution on school children. The advantages of utilizing children for research on the primary etiologic effects of air pollution were first noted by Reid[53] several years ago; Anderson[50] has most recently reaffirmed this view. A major element of concern is that deleterious effects on the respiratory system of very young children may have an effect on the subsequent evolution of the chronic bronchitis syndrome in the adult population.

The relationships of respiratory infections to long-term residence in specific localities have been studied in England by Douglas and Waller.[54] Levels of air pollution, in terms of domestic coal consumption records, were used to classify four groups; the authors include an evaluation of the validity of this method at the end of their report. The histories of 3,866 children born during the first week of March 1946 were followed until 1961, when the children were 15 years of age. Social class composition of these children did not differ significantly from area to area. Measured concentrations for smoke and for SO_2 in 1962 and 1963 were compared with the earlier prediction of pollution intensity based on the coal consumption data, and indicated an overlap for the greater London area of low and moderate groupings; for other areas, the predicted gradient of concentrations was affirmed. Because of the age of the subjects, smoking was apparently not considered in this evaluation. In 1965, 19 percent of the boys and 5 percent of the girls, aged 11 to 13, smoked at least one cigarette a week regularly.[60]

In the Douglas and Waller study, the generation of pollutants in the indoor home environment (e.g., by heating and cooking) was not considered. Interviews were conducted with the mothers when the children were 2 and 4 years of age; information was obtained about upper and lower respiratory illness and recorded hospital admissions for these and other causes. Data about colds, coughs, and hospital admissions were also gathered by school doctors at medical examinations when the children were 6, 7, 11 and 15 years of age. Between the ages of 6½ and 10½, special records for causes of school absence exceeding one week were reviewed with the mothers. A total of 3,131 families remained in the same pollution area throughout the first 11 years of this study. The conclusions of the study were that upper respiratory tract infections were *not* related to the amount of air pollution, but that lower respiratory tract infections were. Frequency and severity of lower respiratory tract infections increased with the amount of air pollution exposure, affecting both boys and girls, and with no differences detectable between children of middle class and working class families. This association was found at each of the examination ages, including age 15. At age 15, persistence of rales and rhonchi (chest noise), possibly the prodrome of adult chronic respiratory disease, was some tenfold less in the very low pollution area, and a factor of two less in the low pollution area than that in the high pollution area. *If* the 1962–1963 measured concentrations for smoke and SO_2 can truly be extrapolated to the 15-year respiratory illness survey, then these British school children experienced increased frequency and severity of lower respiratory diseases in association with annual mean smoke concentrations ranging above 130 $\mu g/m^3$ and SO_2 above 130 $\mu g/m^3$ (0.046 ppm).

The lower respiratory tract findings of Douglas and Waller were confirmed in a study by Lunn *et al.*[16] The patterns of respiratory illness in school children of the age group 5 to 6 have been studied with reference to residence in four areas of Sheffield. Mean daily smoke levels measured in each of four areas ranged from 97 $\mu g/m^3$ in the "low" area to 301 $\mu g/m^3$ in the "high" area; SO_2 concentrations were respectively 123 $\mu g/m^3$ (0.043 ppm) and 275 $\mu g/m^3$ (0.096 ppm) in the two areas, during 1963–1964. Somewhat lower pollution levels were noted the following year, although the gradient between the districts was preserved. Questionnaire to the parents, physical examination, observation for the presence of nasal discharge, examination of the eardrums, and recording of both the forced expiratory volume of 0.75 seconds ($FEV_{0.75}$) and the forced vital capacity (FVC), were completed during each of the summer terms of 1963, 1964, and 1965. Several socioeconomic factors were compared for the various districts; smoking was appropriately disregarded for this age group; internal home environments, or differences in home heating systems, were not reported. The authors conclude that there *is* an association with the levels of atmospheric pollution and chronic *upper* respiratory infections (as indicated by mucopurulent nasal discharge, history of three or more colds yearly, or scarred or perforated eardrum). Further, lower respiratory tract illness (measured by history of frequent chest colds or episodes of bronchitis or pneumonia) was similarly associated. Functional changes, in the form of reduced $FEV_{0.75}$ ratios emerged where there was a past history of pneumonia or bronchitis, of persistent or frequent cough, or of colds going to the chest. There appears, therefore, to be a persistence of respiratory dysfunction, even in the absence of high-pollution extant at the time of the function testing. The lowest "effect" level for smoke and SO_2 is not clearly indicated by this study, but the increased association of "respiratory infections" in school children can be detected for areas whose mean daily figures exceed about 100 μg/m^3 for smoke and 120 μg/m^3 (0.042 ppm) for SO_2.

The exacerbation of acute respiratory illness of school children in Ferrara, Italy, has been studied by Paccagnella *et al.*[57] Air pollution measurements from 1959 through 1964 permitted definition of high, medium, and low zones of pollution. School children in the age range 7 to 12 had daily examinations, and the date of onset of acute respiratory disease was recorded. Although climatic conditions were related to changes in pollutant levels, the onset of acute respiratory disease in the children was *not* correlated significantly with changing air pollution values, except in the poorest socioeconomic area. The pollutant values for this community are, however, lower than those encountered in the British studies previously discussed (20 μg/m^3 to 45 μg/m^3 annual mean). In fact, these are levels which are lower than many rural values in the United States.

Toyama [42] studied two groups of school children, 10 to 11 years old, in Kawasaki, Japan. Sulfation rates at the school in the more polluted area varied from 0.5 to 1.9 and averaged 0.9 mg/100cm^2-day PbO_2; no sulfation rates were given for the school in the area of lower pollution. Dustfall was considerably less in the area of lower pollution (ranging from about 5 tons/km^2-mo. to 15 tons/km^2-mo.)* than in the more polluted area (ranging from about 15 tons/km^2-mo. to 70 tons/km^2-mo.). The children from the more polluted area had a higher frequency of nonproductive cough, irritation of the upper respiratory tract, and increased mucus secretion. Whether the effect was due to oxides of sulfur or particulate matter cannot be determined from this study.

A study by Manzhenko [58] of upper respiratory tract conditions in school children in Irkutsk is difficult to relate to the Sheffield study. However, the higher incidence of respiratory tract conditions and the undefined abnormal X-ray findings in these children's lungs are disturbing evidence of the possibility of an association between serious respiratory disease and residence in a polluted community.

4. Studies of Pulmonary Function

Holland *et al.*[43] report decreased performance of the $FEV_{1.0}$ test in London and British rural outside telephone workers compared with their American counterparts. For both groups the FEV was further decreased in relation to smoking intensity. FEV differences within the United Kingdom workers (i.e., London versus rural) could also be related to the sulfur dioxide concentrations accompanying the particulate levels. A more detailed discussion of the study appears in Section C–4.

Toyama [42] reported measurements of peak flow rate and total vital capacity performance in Japanese school children in areas of differing air pollution, measured monthly; fluctuations were observed in the mean peak flow rates of children attending schools and living in polluted industrial areas; and the variations were smaller for children in clearer areas. Total vital capacity was not significantly different between pupils of the various schools. There was a substantial difference in peak flow rates between the two school districts at times of highest pollution. When pollution values were lowest, the differences were less. The lowest values related to dustfall measurements of 60 tons/km^2-mo.* and daily sulfur dioxide levels equivalent to 20 μg/cm^2 (lead candle measurement).

In Osaka, Watanabe [20] studied the peak flow rate and vital capacity performances by the children of schools enduring differing air pollutant concentrations. It was noted that individual peak flow rates were more markedly decreased in the winter months (September to December, 1963) for the school in the highly polluted area than for the school in the low pollution area. Daily mean concentrations of both dustfall and sulfur dioxide concentrations were twice as great in the polluted area as in the unpolluted area.

In a comparison by Prindle *et al.*[59] of pulmonary function and other parameters in two Pennsylvania communities with widely different air pollution levels, average airway resistance and specific airway resistance were measured in persons 30 years of age and older. These measurements were performed at several stations in each of the communities and indicated differences between the high-pollution and low-pollution communities which were probably related to the sixfold greater dustfall. However, smoking and occupation were not accounted for.

---

*In the United States, dustfall is measured in tons/mi^2-mo.

*In the United States, dustfall is measured in tons/mi^2-mo.

In the paper by Lunn *et al.*,[56] Sheffield school children were shown to have reduced FEV$_{0.75}$ and FVC ratios in the area of highest pollution. The measurements were made during the summer, when pollution levels were low and apparent incidence of acute respiratory infection was diminished, suggesting, in contrast to the Japanese studies referred to above, that there may be persistence of the respiratory function deterioration in relation to residence in the area of high pollution; mean daily averages measured at a single station were: smoke, 300 μg/m^3; SO$_2$, 275 μg/m^3 (0.096 ppm).

The studies relating morbidity and deterioration in pulmonary function to particulate levels cover effects which are also included in Level III of the World Health Organization's "guides to air quality."[23]

5. Studies of Panels of Bronchitis Patients

Lawther[60] related several episodes of acute urban pollution to worsening of condition in a group of bronchitic patients, well studied in a registry at St. Bartholomew's hospital. Changes in their symptomatology were recorded in a daily diary and acute worsening in significant numbers of the group was associated with daily rises in air pollution above 300 μg/m^3 of smoke and 600 μg/m^3 (0.21 ppm) of sulfur dioxide. Figure 11–8 shows graphically the effects observed on 29 bronchitic patients of high pollution levels in January 1954.

Angel *et al.*[62] reviewed the occurrence of new respiratory symptoms in men, working in factories and in offices, most of whom had prior evidence of chronic bronchitis. The study group of 85 men observed through the winter of 1962–1963 was selected from a group of 1,000 men, age 30 through 59, without apparent classification of either smoking patterns or possible occupational or residential exposure differences. Increased sputum production, deterioration of pulmonary function performance (FEV$_{1.0}$), and the more frequent occurrence of respiratory symptoms classified as "upper" (coryza, influenza, and acute respiratory disease), and "lower" (chest colds, bronchitis, wheezy attacks, pneumonia) were all associated with increases in both smoke and sulfur dioxide concentrations. There was frequently difficulty in defining an exacerbation of disease in those individuals already experiencing chronic bronchitis. During this period, illness peaks (attack rate) may have occurred with weekly mean concentrations of smoke exceeding 400 μg/m^3 and of sulfur dioxide exceeding 460 μg/m^3 (0.16 ppm); weekly mean concentrations were calculated using the highest daily mean occurring each week at each of 13 locations in the area.

Bierstecker[63] surveyed male municipal employees in Rotterdam for symptoms of bronchitis and for peak flow meter performance, and reviewed years of residence in Rotterdam versus years of residence in a nonurban environment as well as smoking habits. The individuals with bronchitis symptoms were matched with individuals without such symptoms but with similar age and background. Significant differences related to heavy cigarette smoking, but no reliable statistical indication of an effect of urban or nonurban residence in the production of symptoms was detectable. Since particulate levels in Rotterdam are very low, the range of difference between particulate levels in Rotterdam and the nonurban environment may have been close to minimal.

Fletcher *et al.*[64] followed 1,136 working men aged 30 to 59 in West London by surveys at 6-month intervals. The surveys included collection and measurement of morning sputum volume, FEV, and respiratory symptom questionnaires. While expected patterns of decline in lung function with age occurred and this was the most rapid in cigarette smokers with low lung fuction to start with, an unexpected finding was a decrease in sputum volume in men with constant smoking habits. This was most con-

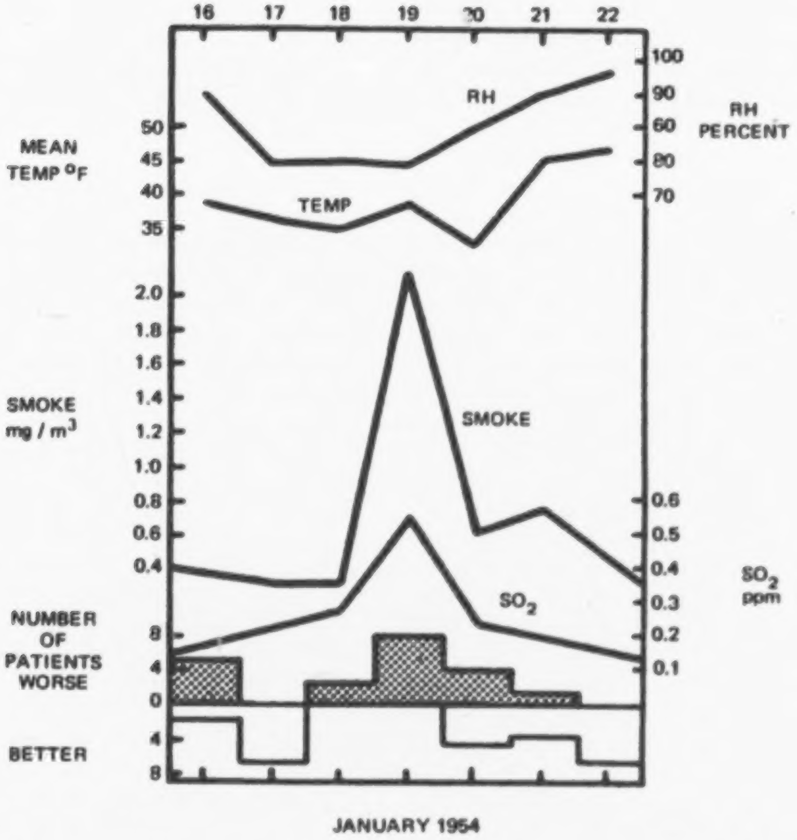

Figure 11–8. Effect on Bronchitic Patients of High Pollution Levels (January 1954).[60,61] (The figure represents the effect on bronchitic patients of increased pollution levels; patients stated whether they regarded their condition as "worse" or better".)

sistent in the winter samples, but showed a significant trend in samples taken together as well as in the winter samples. (Trend data are based on 825 men who attended at least nine periodic examinations.) The authors felt this was possibly due to a decline in air pollution in London. They presented data on trends in SO_2 and black suspended matter which indicated that there is a consistent downward trend in particulate pollution (highly significant) but a less steady decline in SO_2.

This unique set of data could mean that with a decrease of smoke pollution (yearly mean) from 140 $\mu g/m^3$ to 60 $\mu g/m^3$, there is an associated decrease in mean sputum volume during the first morning hour from about 1.5 ml to about 0.75 ml. A contribution of the decrease in SO_2 is less likely than that of the decrease in smoke. Possible changes in cigarette tars, in methods of smoking and in techniques for collecting a sample, or other factors, could have influenced this result.

1st hour sputum volume—In men of constant smoking habits

	1961	*1962*	*1963*	*1964*	*1965*	*1966*
Summer	1.65	0.83	0.93	0.93	0.83	0.72
Winter	1.38	1.27	1.20	1.13	0.70	......

D. SUMMARY

This chapter reviews epidemiologic studies of the relationship between pollutant concentrations and their effects on health. Indices varying from disturbance of lung function to death are considered. Concentration measurements of pollutants are sometimes available for the period during which the indicators were under review, and in these cases it is possible to state pollutant levels at which an effect is noted. However, such values cannot be taken to mean that effects will not be noted at lower concentrations. Most studies involving long-term effects using a geographic area comparison will use the cleanest area as the "control" area against which mortality and morbidity differentials are to be found. This necessarily assumes that this "cleanest" area itself does not have any effects from its level of pollution. Conversely, some studies have compared areas with very low particulate loading or with very limited ranges of differences. It is not possible to demonstrate any relationship under conditions of minimum values or minimal differences. Table 11-6 summarizes the epidemiologic studies reviewed according to the several indices.

From the material reviewed in this chapter, a selection has been made of data from those studies which furnish the best quantitative information that we have available at the present time. Levels are given in the measurement system used in the original observations, since conversion from one method to another is not recommended. Attention should be given to the difference between British "smoke" and American "suspended particulate" measurements. Both are given in micrograms per cubic meter of air, but they are not identical. Limited data indicate that the American values may be higher in the same situation. In making use of these data, attention must also be given to the averaging time which was employed in the original observation. Long-term averages are, of necessity, considerably lower than selected high daily means in the same locations.

British studies of acute episodes of increased pollution show excess deaths occurring at smoke levels from 750 $\mu g/m^3$ to 2,000 $\mu g/m^3$. High SO_2 levels are, of course, concurrently present. The excesses of mortality are always accompanied by a very large increase in illness, mainly exacerbations of chronic conditions. Similar but less spectacular episodes in New York City have been associated with smoke shade levels of 5-6 coh units.

Lawther, in reviewing a long series of observations on the condition of bronchitic patients, estimated that they tended to become worse when daily levels of smoke exceeded 300 $\mu g/m^3$ with SO_2 over 600 $\mu g/m^3$. Angel *et al.* made somewhat similar observations with smoke above 400 $\mu g/m^3$ and SO_2 over 460 $\mu g/m^3$.

Winkelstein found in Buffalo that increases in the mortality rate were significantly linked to higher levels of suspended particulate pollution. His studies showed that mortality from all causes, from chronic respiratory diseases, and from gastric carcinoma increased from the lowest of his five levels of pollution (less than 80 $\mu g/m^3$) through the three higher ranges, after the effects of socioeconomic status had been considered. Zeidberg found in Nashville significant increases in all respiratory deaths at soiling levels over 1.1 cohs annual average. Neither of these studies took smoking habits into account, and the Nashville study only partially allowed for socioeconomic factors.

Studies of illness in relation to residence in more- and less-polluted areas contribute additional information. Fletcher *et al.* noted a proportional decline in the production of morning sputum in chronic bronchitics in West London from 1961 to 1966 as smoke pollution in their residence areas declined from 140 $\mu g/m^3$ annual mean. Douglas and Waller found an increase in frequency and severity of lower respiratory illness at smoke and SO_2 levels over 130 $\mu g/m^3$ annual average. The Sheffield study by Lunn *et al.*[2] shows similar differences occurring with some morbidity measures between about 100 $\mu g/m^3$ and 200 $\mu g/m^3$ of smoke, and for others between 200 $\mu g/m^3$ and 300 $\mu g/m^3$ annual average.

A study of British workmen found increased respiratory illness absence in areas with smoke levels in excess of 200 $\mu g/m^3$.

Physiologic studies of lung function have also been made in both adults and children. On the basis of present limited knowledge it appears that the alterations found may be both temporary and permanent. The observations now available relate to long-term residence in a given area. The Sheffield

Table 11-6.—SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter $\mu g/m^3$	Smoke or black suspended matter (B. S. M.) $\mu g/m^3$	(in brackets) or COH dustfall tons/mi²-mo	SO_2 $\mu g/m^3$ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
A. Acute episodes—Mortality:						
1. London, winter 1958–59.	------	rise of 200	------	rise of 75	Correlation of daily mortality (all causes) and daily bronchitis mortality with B.S.M. Significant positive association of SO_2 and deaths.	11
London, winter 1958–59.	------	1000	------	715	Increase in mortality (all causes)	12
London, winter 1958–59.	------	750	------	715	Re-analysis of data of reference 4	13
2. London, fog episodes, 1954, 1955, 1956, 1957.	------	2000	------	1200	Bronchitis death rate increased, other causes rise also.	8,9
London, fog episode 1962.	------	2000	------	1145	Death rates of elderly rose; greater proportionate rise in bronchitis deaths.	10
3. New York, November, 1953.	------	-----------	[5]	2460	Excess deaths all age groups; lag effect noted.	14
4. New York, December, 1962.	------	-----------	------	-----------	No excess deaths	15
New York, December, 1962.	------	-----------	[6]	2145	Excess deaths in 45–64 age group and over 65 age group.	16
5. New York, January, 1963.	------	-----------	[6]	1715	Peak in death rate superimposed on high deaths due to influenza.	16
6. Detroit, September, 1952.	>200 3 days	-----------	------	2860 (max)	Excess infant mortality and cancer mortality.	19
7. Osaka, December, 1962.	1000 (sic)	-----------	------	285	Sixty excess deaths	20
B. Acute episodes—Morbidity:						
1. New York, November, 1953.	------	-----------	[3]	715	Increased upper respiratory infection and cardiac disease morbidity.	21
2. New York, November, 1966.	------	-----------	------	-----------	Increased emergency visits for bronchitis and asthma; analysis complicated by Thanksgiving holiday.	22
C. Long-term air pollution—Day to day variations in mortality and morbidity:						
1. London winters, 1958–59, 1959–60.	------	>500	------	>400	Increases in mortality and morbidity appear positively associated with fluctuations in air pollution.	12

Table 11–6 (continued).—SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter μg/m³	Smoke or black suspended matter (B. S. M.) μg/m³	(in brackets) or COH dustfall tons/mi²-mo	SO_2 μg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
2. New York City 1962–64.	------	-----------	------	-----------	Some periods of high air pollution appear to be related to air pollution; relationship not clear during other periods of high air pollution.	16
3. New York City, housing project.	------	-----------	------	-----------	Cough symptoms related to rise in air pollution in general; eye irritation related to rise in SO_2.	22
D. Long-term air pollution—Geographic variations in mortality:						
1. U.K., 214 areas, 1955–59.	------	200	------	200	Excess bronchitis mortality. Social index also important. Pollutant concentrations not measured in same period as mortality.	26
2. U.K., 1950–54 lung cancer; 1950–53 bronchitis, pneumonia; 1954–57 sampling network data.	n.a.	-----------	------	n.a.	Lung cancer mortality, bronchitis, and pneumonia mortality in males and bronchitis in females, strongly correlated with smoke density. Cancer of stomach and intestine correlates for some areas.	27–31
U.K., 1950–54 lung cancer; 1950–53 bronchitis, pneumonia; 1954–57 sampling network data.	------	-----------	------	-----------	Smoke, occupational, and population parameters relate to lung cancer mortality. Vanadium contributes to lung, female cancer, pneumonia mortalities. Pollutant measurements do not parallel time period of mortality studies.	
3. Eston, England.	------	160	------	115	Positive association between the degree of air pollution and the incidence of both bronchitis and lung cancer.	33
4. Salford, U.K., 3 wards, August, 1957, through.	------	"High": winter [illegible]80, summer 270 "Low": winter 450, summer 170.	------	"High" winter 750, summer 270 "Low": winter 360, summer 150.	High rate of bronchitis and lung cancer mortality. Death from all causes increased in "high" compared to "low" pollution wards.	34
5. Buffalo, N.Y., areas July, 1961–June, 1963.	100	-----------	------	[SO_3 0.45]	Positive correlation of pollutant levels with chronic respiratory disease deaths, and deaths from all causes. Mortality rates for gastric cancer 2 times higher in high particulate areas as compared to low particulate area.	35–37

Table 11-6 (continued).—SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter μg/m³	Smoke or black suspended matter (B. S. M.) μg/m³	COH (in brackets) or [illegible]fall tons/mi²-mo	SO₂ μg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
					Air pollution levels did not relate to deaths due to cancer of bronchus, trachea or lung. Mortality effects also seen in area with 80 μg/m³ to 100 μg/m³.	
6. Nashville, Tenn., 1940–60.	------	-----------	[>1.1]	30	"High" pollution related to increased respiratory disease mortality. Lung and bronchial cancer, bronchitis, and emphysema mortality not clearly related.	38,39
E. Long-term air pollution—Geographic variations in morbidity:						
1. Genoa, Italy	------	-----------	------	-----------	Frequency of bronchitis significantly associated with SO₂, not with suspended matter of dustfall.	41
2. Japan	------	n.a.	------	n.a.	Gradient of respiratory disease morbidity from industrial to rural sites.	42
3. U.K.-U.S. Comparison.	U.S. 100	U.K. 200	------	U.S. 12–30 U.K. 60–300	Increase of smoke concentration from 100 μg/m³ to 200 μg/m³ and equivalent SO₂ increase leads to higher risk to older men of poorer lung function and increased chronic respiratory disease.	43
		(Levels not directly comparable)				
4. U.K	------	-----------	[11.6] (1 yr. av.)	[SO₃ 1.3]	Differences in area respiratory morbidities attributed by authors to difference in air pollution.	44
5. Canada, 4 cities.	Inadeq.	-----------	------	Inadeq.	Increased severe bronchitis, poor lung function performance in "dirty" cities versus "clean" cities.	45
6. Berlin, N. Hampshire, Chilliwack, B.C.	------	-----------	34.9	[SO₃ 0.4] [SO₃ 0.05]	No observable differences	46–48
7. Nashville, Tenn.	------	-----------	------	-----------	Cardiovascular disease directly correlated with aerometric data. No increased respiratory disease morbidity.	49
F. Long-term air pollution—Incapacity for work:						
1. U.S	100–190	-----------	------	-----------	No correlation of absence in excess of 7 days with suspended particles. Significant correlation of absence with "suspended particulate sulfate."	50

Table 11-6 (continued).—SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

	Health Index	Suspended particulate matter µg/m³	Smoke or black suspended matter (B. S. M.) µg/m³	(in brackets) or COH dustfall tons/mi²-mo	SO_2 µg/m³ daily average bracketed values are sulfation rates in mg/100 cm²-day	Findings	Reference
	2. Salford, U.K....	------	>1000 (2 consecutive days)	------	-----------	Bronchitis caused absence greater than expected by factor of 2 in four of five episodes.	34
	3. Greater London, U.K.	------	100–200	------	150–250	Lowest bronchitis inception rates..	51
			400		400	Highest bronchitis inception rates. Significant correlation of bronchitis incapacity with smoke and SO_2 for all age groups, and men 35–54 and 55–59.	
	4. New York, N.Y.	------	-----------	[2.7] (7 yr. mean).	540 (7 yr. mean).	No strong relation between respiratory illness absence and COH or SO_2.	52
	Studies of children:						
G.	1. U.K., 1946–61.	>130	-----------	------	>130	Increased frequency and severity of lower respiratory diseases in school children.	54
	2. Sheffield, U.K., 1963–65.	100	-----------	------	>120	Increased association of "respiratory infections" for school children age 5–6.	56
	3. Ferrara district, Italy, 1959–64.	n.a.	-----------	------	n.a.	No correlation of onset of acute respiratory illness to changing pollutant levels except in poorest socioeconomic area.	57
	4. Japan........	------	-----------	60	[SO_3 0.02]	Total vital capacity of children in polluted and nonpolluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	42
	5. Japan........	------	-----------	------	-----------	Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	20
	6. Irkutsk, U.S.S.R., time period not definite.	n.a.	-----------	------	n.a.	Possible relation of respiratory disease and residence in area of pollution.	58
H.	Studies of pulmonary function:						
	1. U.K.-U.S. Comparison.	------	-----------	------	-----------	$FEV_{1.0}$ performance of U.K. workers decreased compared to U.S. counterparts.	43
	2. Japan........	------	-----------	------	-----------	Total vital capacity of children in polluted and nonpolluted areas the same. Fluctuations in mean peak flow rates larger for children in polluted areas than in less polluted areas.	42

Table 11-6 (continued).—SUMMARY TABLE OF EPIDEMIOLOGICAL STUDIES

Health Index	Suspended particulate matter $\mu g/m^3$	Smoke or black suspended matter (B. S. M.) $\mu g/m^3$	(in brackets) or COH dustfall tons/mi^2-mo	SO$_2$ $\mu g/m^3$ daily average bracketed values are sulfation rates in mg/100 cm^2-day	Findings	Reference
3. Japan	------	-----------	------	-----------	Peak flow rates decreased more in winter for children in polluted areas than in less polluted areas.	20
4. Pennsylvania, 2 communities.	------	-----------	------	-----------	Possible relation of dustfall and SO$_2$ differences in average and specific airway resistance of subjects in the two communities.	59
5. Sheffield, U.K.	------	100	------	275	Reduced FEV$_{0.75}$ and FVC in areas of highest pollution. Possible persistence of respiratory function deterioration related to residence in area of high pollution.	56
I. Studies of panels of bronchitic patients:						
1. London, November, 1955–May, 1956.	300	-----------	------	>600	Acute worsening of symptoms in bronchitis patients.	60
2. U.K., October, 1962–April, 1963.	400	-----------	------	460	Possible increase in respiratory disease attack rates.	62
3. Rotterdam, Netherlands.	------	n.a.	------	n.a.	No indication of residence effect on bronchitis symptoms.	63
4. London	------	140 $\mu g/m^3$ declining to 60 $\mu g/m^3$.	------	200 $\mu g/m^3$ declining to 160 $\mu g/m^3$.	Decrease in morning sputum volume with decreasing air pollution levels in bronchitis patients under observation during 6 years.	64

study shows reduced pulmonary function in the children in the most polluted area, i.e., where smoke concentration is above 300 $\mu g/m^3$. Studies in Japan show a decrease in pulmonary function in school children living in areas of high dustfall as compared with those living in low dustfall areas. In Osaka the dustfall levels were 6.5 gm/m^2-month and 13.3 gm/m^2-month.

The analyses of the numerous epidemiological studies discussed clearly indicate an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain a reservoir of individuals with impaired health who are prime targets for the effects of elevated levels of particulate matter and sulfur oxides. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these small changes cannot be detected statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily detected in major urban areas.

It is believed that, for the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can detect with confidence the small changes in daily mortality which are associated with functions in pollution concentrations. Unfortunately, only limited analysis has thus far been made, and this has been attempted only in London and in New York.

The association between longer-term community exposures to particulate matter for respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status, must still be considered consequential.

E. REFERENCES

1. Hill, A. B. "The Environment and Disease: Association or Causation." Proc. Royal Soc. Med., Vol. 58, pp. 295–300, 1965.
2. Cassel, E. G., Walter, E. W., Mountain, J. D., Diamond, J. R., Mountain, I. M., and McCarroll, J. R. "Reconsiderations of Mortality as a Useful Index of the Relationship of Environmental Factors to Health." Am. J. Pub. Health, Vol. 58, pp. 1653–1657, 1968.
3. Anderson, D. O. "The Effects of Air Contamination on Health, Part I." Canad. Med. Assoc. J. 97:528–536, Sept. 2, 1967.
4. Firket, J. "Fog along the Meuse Valley." Trans. Faraday Soc. 32:1192–1197, 1936.
5. Firket, J. "The Cause of the Symptoms Found in the Meuse Valley during the Fog of December, 1930." Bull. Roy. Acad. Med. (Belgium) 11:683–739, 1931.
6. Brasser, L. J., Joosting, P. E., and von Zuilen, D. "Sulfur Dioxide—to What Level is it Acceptable?" Report G-300. Research Institute for Public Health Engineering, Delft, Netherlands. July 1967. (Originally published in Dutch, Sept. 1966.)
7. Joosting, P. E. "Air Pollution Permissibility Standards Approached from the Hygienic Viewpoint." Ingenieur, *79*(50):A739–A747, 1967.
8. Gore, A. T. and Shaddick, C. W. "Atmospheric Pollution and Mortality in the County of London." Brit. J. Prevent. Soc. Med. 12:104–113, 1958.
9. Burgess, S. E. and Shaddick, C. W. "Bronchitis and Air Pollution." Roy. Soc. Health J. 79:10–24, 1959.
10. Scott, J. A. "The London Fog of December 1962." Med. Officer 109:250–252, 1963.
11. Martin, A. E. and Bradley, W. "Mortality, Fog and Atmospheric Pollution." Monthly Bull. Ministry Health 19:56–59, 1960.
12. Martin, A. E. "Mortality and Morbidity Statistics and Air Pollution." Proc. Roy. Soc. Med. 57:969–975, 1964.
13. Lawther, P. J. "Compliance with the Clean Air Act: Medical Aspects." J. Inst. Fuels (London) 36:341–344, 1963.
14. Greenburg, L., Jacobs, M. B., Drolette, B. M., Field, F., and Braverman, M. M. "Report on an Air Pollution Incident in New York City, November 1953." Public Health Repts. 77:7–16, 1962.
15. Greenburg, L., Erhardt, C., Field, F., Reed, J. I., and Seriff, N. S. "Intermittent Air Pollution Episodes in New York City, 1962." Public Health Repts. 78:1061–1065, 1963.
16. McCarroll, J. and Bradley, W. "Excess Mortality as an Indicator of Health Effects of Air Pollution." Am. J. Public Health 56:1933–1942, 1966.
17. Glasser, M., Greenburg, L., and Field, F. "Mortality and Morbidity during a Period of High Levels of Air Pollution, New York, November 23–25, 1966." Arch. Environ. Health 15:684–694, 1967.
18. Leonard, A. G., Crowley, D., and Belton, J. "Atmospheric Pollution in Dublin during the Years 1944 to 1950." Royal Dublin Soc. Sci. Proc. 25:166–167, 1950.
19. "Report of the International Joint Commission, United States and Canada, on the Pollution of the Atmosphere in the Detroit River Area." International Joint Commission (United States and Canada). 1960. p. 115.
20. Watanabe, H. "Air Pollution and its Health Effects in Osaka, Japan." Preprint. (Presented at the 58th Annual Meeting, Air Pollution Control Association, Toronto, Canada, June 20–24, 1965.)
21. Greenburg, L., Field, F., Reed, J. I., and Erhardt, C. L. "Air Pollution and Morbidity in New York City." J. Amer. Med. Assoc., Vol. 182, pp. 161–164, 1962.

22. McCarroll, J. R., Cassel, E. G., Walter, E. W., Mountain, J. D., Diamond, J. R., and Mountain, I. R. "Health and the Urban Environment. V. Air Pollution and Illness in a Normal Urban Population." Arch. Environ. Health, Vol. 14, pp. 178–184, 1967.

23. "Atmospheric Pollutants." Technical report series No. 271. World Health Organization, Geneva, Switzerland. 1964.

24. Kurland, L. T. Personal Communication. Nov. 1, 1968.

25. Markush, R. E. "National Chronic Respiratory Disease Mortality Study. 1. Prevalence and Severity at Death of Chronic Respiratory Disease in the United States, 1963." J. Chron. Dis., Vol. 21, pp. 129–141, 1968.

26. Buck, S. F. and Brown, D. A. "Mortality from Lung Cancer and Bronchitis in Relation to Smoke and Sulfur Dioxide Concentration, Population Density, and Social Index." Research paper No. 7. Tobacco Research Council, London, England. 1964.

27. Stocks, P. "Statistics of Cancer of the Lung." J. Faculty Radiologists 6:166–173, 1955.

28. Stocks, P. "Air Pollution and Cancer Mortality in Liverpool Hospital Region and North Wales." Intern. J. Air Pollution 1:1–13, 1958.

29. Stocks, P. "Cancer and Bronchitis Mortality in Relation to Atmospheric Deposit and Smoke." Brit. Med. J. 1:74–79, Jan. 10, 1959.

30. Stocks, P. and Davies, R. I. "Epidemiological Evidence from Chemical and Spectrographic Analyses that Soil is Concerned in the Causation of Cancer." Brit. J. Cancer 14:8–22, 1960.

31. Stocks, P. "On the Relations between Atmospheric Pollution in Urban and Rural Localities and Mortality from Cancer, Bronchitis, and Pneumonia, with Particular Reference to 3:4 Benzopyrene, Beryllium, Molybdenum, Vanadium, and Arsenic." Brit. J. Cancer 14:397–418, 1960.

32. Anderson, D. O. "The Effects of Air Contamination on Health: Part III." Canad. Med. Assoc. J. 97:802–806, Sept. 23, 1967.

33. Wicken, A. J., and Buck, S. F. "Report on a Study of Environmental Factors Associated with Lung Cancer and Bronchitis Mortality in Areas of North East England." Tobacco Research Council, London, Research Paper 8, 1964.

34. Burn, J. L. and Pemberton, J. "Air Pollution, Bronchitis, and Lung Cancer in Salford." Intern. J. Air Water Pollution 7:5–16, 1963.

35. Winkelstein, W. "The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Man." Arch. Environ. Health 14:162–169, 1967.

36. Winkelstein, W. "The Relationship of Air Pollution and Economic Status to Total Mortality and Selected Respiratory System Mortality in Men (II. Oxides of Sulfur)." Arch. Environ. Health 16:401–405, 1968.

37. Winkelstein, W. "The Relationship of Air Pollution to Cancer of the Stomach." Arch. Environ. Health. 18:544–547, 1969.

38. Zeidburg, L. D., Hagstrom, R. M., Sprague, H. A., and Landau, E. "Nashville Air Pollution Study. VII. Mortality from Cancer in Relation to Air Pollution." Arch. Environ. Health 15:237–248, 1967.

39. Zeidberg, L. D., Horton, R. J. M., and Landau, E. "The Nashville Air Pollution Study. V. Mortality from Diseases of the Respiratory System in Relation to Air Pollution." Arch. Environ. Health 15:214–224, 1967.

40. Sprague, H. A. and Hagstrom, R. M. "The Nashville Air Pollution Study: Mortality Multiple Regression." Arch. Environ. Health. 18:503–507, 1969.

41. Petrilli, F. L., Agnese, G., and Kanitz, S. "Epidemiology Studies of Air Pollution Effects in Genoa, Italy." Arch. Environ. Health 12:733–740, 1966.

42. Toyama, T. "Air Pollution and its Health Effects in Japan." Arch. Environ. Health 8:153–173, 1964.

43. Holland, W. W., Reid, D. D., Seltser, R., and Stone, R. W. "Respiratory Disease in England and the United States. Studies of Comparative Prevalence." Arch. Environ. Health 10:338–345, 1965.

44. Holland, W. W. and Reid, D. D. "The Urban Factor in Chronic Bronchitis." Lancet 1:445–448, 1965.

45. Bates, D. V. "Air Pollution and Chronic Bronchitis." Arch. Environ. Health 14:220–224, 1967.

46. Ferris, B. G., Jr. and Anderson, D. O. "The Prevalence of Chronic Respiratory Disease in a New Hampshire Town." Am. Rev. Respirat. Diseases 86:165–177, 1962.

47. Anderson, D. O., Ferris, B. G., and Zickmantel, R. "Levels of Air Pollution and Respiratory Disease in Berlin, New Hampshire." Am. Rev. Respirat. Diseases 90:877–887, 1964.

48. Anderson, D. O. and Ferris, B. G. "Air Pollution Levels and Chronic Respiratory Disease." Arch. Environ. Health 10:307–311, 1965.

49. Zeidburg, L. D., Prindle, R. A., and Landau, E. "The Nashville Air Pollution Study. III. Morbidity in Relation to Air Pollution." Am. J. Public Health 54:85–97, 1964.

50. Dohan, F. C. "Air Pollutants and Incidence of Respiratory Disease." Arch. Environ. Health 3:387–395, Oct. 1961.

51. "Report on an Enquiry into the Incidence of Incapacity for Work: Part II. Incidence of Incapacity for Work in Different Areas and Occupations." Ministry of Pensions and National Insurance, London, England. Her Majesty's Stationery Office, London, England. 1965.

52. Verma, M. P., Schilling, F. J., and Becker, W. H. "Epidemiological Study of Illness Absences

in Relation to Air Pollution." Arch. Environ. Health. 18:536–543, 1969.

53. Reid, D. D. "Air Pollution and Respiratory Disease in Children." In: Bronchitis, Second International Symposium, Groningen, The Netherlands, April 22–24, 1964.

54. Douglas, J. W. B. and Waller, R. E. "Air Pollution and Respiratory Infection in Children." Brit. J. Prevent. Soc. Med. 20:1–8, 1966.

55. Holland, W. W. and Elliott, A. "Cigarette Smoking, Respiratory Symptoms, and Antismoking Propaganda: an Experiment." Lancet 1:41–43, 1968.

56. Lunn, J. E., Knowelden, J., and Handyside, A. J. "Patterns of Respiratory Illness in Sheffield Infant School Children." Brit. J. Prevent. Soc. Med. 21:7–16, 1967.

57. Paccagnella, B., Pavanello, R., and Pesarin, F. "The Immediate Effects of Air Pollution on the Health of School Children in Some Districts of Ferara." Arch. Environ. Health. 18:495, 502, 1969.

58. Manzhenko, E. G. "The Effect of Atmospheric Pollution on the Health of Children." Hyg. Sanit. 31:126-128, 1966.

59. Prindle, R. A., Wright, G. W., McCaldin, R. O., Marcus, S. C., Lloyd, T. C., and Bye, W. E. "Comparison of Pulmonary Function and Other Parameters in Two Communities with Widely Different Air Pollution Levels." Am. J. Public Health 53:200–218, 1963.

60. Lawther, P. J. "Climate, Air Pollution, and Chronic Bronchitis." Proc. Roy. Soc. Med. 51: 262–264, 1958.

61. Waller, R. E. and Lawther, P. J. "Some Observations on London Fog." Brit. Med. J. 4952:1356–1358, Dec. 3, 1955.

62. Angel, J. H., Fletcher, C. M., Hill, I. D., and Tinker, C. M. "Respiratory Illness in Factory and Office Workers." Brit. J. Diseases Chest 59:66–79, 1965.

63. Biersteker, K. "Air Pollution and Smoking as Cause of Bronchitis among 1,000 Male Municipal Employees in Rotterdam (The Netherlands). Arch. Environ. Health. 18:531–535, 1969.

64. Fletcher, C. M., Tinker, B. M., Hill, I. D., and Speizer, F. E. "A Five-Year Prospective Field Study of Chronic Bronchitis." Preprint. (Presented at the 11th Aspen Conference on Research in Emphysema June 1968.)

Chapter 12

SUMMARY AND CONCLUSIONS

Table of Contents

Page

A. SUMMARY 181
- 1. General 181
- 2. Effects on Health 182
- 3. Effects on Climate Near the Ground 184
- 4. Effects on Visibility 184
- 5. Effects on Materials 185
- 6. Economic Effects of Atmospheric Particulate Matter 186
- 7. Effects on Vegetation 186
- 8. Effects on Public Concern 187
- 9. Suspended Particles as a Source of Odor 187

B. CONCLUSIONS 187
- 1. Effects on Health 188
- 2. Effects on Direct Sunlight 189
- 3. Effects on Visibility 189
- 4. Effects on Materials 189
- 5. Effects on Public Concern 189

C. RESUME 189

Chapter 12

SUMMARY AND CONCLUSIONS

A. SUMMARY

1. General

The particulate matter commonly found dispersed in the atmosphere is composed of a large variety of substances. Some of these—fluorides, beryllium, lead, asbestos, for example—are known to be directly toxic, although not necessarily at levels routinely found in the atmosphere today. The evidence suggests that there may very well be others whose toxic effects have not yet been recognized. To evaluate fully the effects on health and welfare of the presence of each of these substances in the air requires that they be given individual attention, or attention as classes of similar substances. Such evaluations will be made in separate documents.

This document considers the effects on man and his environment of undifferentiated particulate matter. These effects oftentimes are produced by a combination of particulate and gaseous pollutants, the contributions of which are difficult to distinguish. Moreover, laboratory studies have shown that a combination of particulates and gases may produce an effect that is greater than the sum of the effects caused by these pollutants individually.

Particles in the atmosphere, whatever their individual characteristics, exhibit a number of similar properties, which are for the most part dependent on the particle size. Most of the available studies on the effects of particulate air pollution, however, do not specify particle size, and this document is limited to treating particulate matter as a whole, and to considering the effects which are generally associated with the presence of particles in the air.

Particulate air pollution, as used in this document, refers to any matter dispersed in the air, whether solid or liquid, in which the individual particles are larger than small molecules but smaller in diameter than 500μ. (One μ in one millionth of a meter.) Particles in this size range stay in the air anywhere from a few seconds to several months.

Generally speaking, particles smaller than 1 μ in diameter originate in the atmosphere principally through condensation and combustion, while larger particles, with the exception of rain, snow, hail, and sleet, arise principally from comminution. Particles larger than 10 μ in diameter result from mechanical processes such as wind erosion, grinding and spraying, and the pulverizing of materials by vehicles and pedestrians. Particles between 1 μ and 10 μ in diameter usually include local soil, process dusts and combustion products from local industries, and, for maritime locations, sea salt. Combustion products and photochemical aerosols make up a large fraction of the particles in the range 0.1 μ to 1 μ in diameter, and, although particles below 0.1 μ in diameter have not been extensively identified chemically, the typical urban increase over natural levels of particles in this size range seems to be entirely due to combustion.

Particles of a size less than 0.1 μ in diameter are characterized by random motions produced by collisions with gas molecules. They are highly concentrated, move rapidly, collide frequently, and through sorption and nucleation of gas molecules and adhesion with other particles grow larger quickly. Particles larger than 1 μ have significant settling velocities, and their motions may deviate significantly from the motion of the air.

Measurements of dustfall are commonly used to indicate the mass concentration of the settleable portion of particulate air pollution. Typical values for cities are 10 to 100 tons/mile^2-month; as high as 2,000 tons/mile^2-month have been measured in the vicinity of especially offensive sources. Levels of dustfall have apparently declined in some American cities, and dustfall measurements are probably not useful as an index of overall particulate air pollution. However, dustfall itself constitutes a nuisance, and its measurement can be used as an index of the dirtiness of air pollution.

Several methods are available for measuring suspended particulate concentrations. The most commonly used device is the high-volume sampler, which consists essentially of a blower and a filter, and which is usually operated in a standard shelter to collect a 24-hour sample. The sample is weighed to determine concentration, and is usually analyzed chemically. The hi-vol is considered a reliable instrument for measuring the weight of total particulate matter. Chemical analysis of the hi-vol sample, however, may be limited: the filter material may contaminate the sample; different substances in the sample may react with each other; and losses may occur through volatilization of material. Tape samplers, which collect suspended particulate matter on filters and analyze the sample optically, are also in common use. While these samplers are inexpensive and rugged, they yield data which cannot always be easily interpreted in terms of particulate mass concentration. Other techniques available for measuring particulate pollution include optical systems, which provide an indication of concentration without requiring that a sample be taken.

The averaging time used for measuring suspended particulates is not as significant a factor as it is for gaseous pollutants. The basic unit of time is 24 hours. Values taken over this period may be combined into weekly, monthly, seasonal, and annual means as required. The relationships between daily and other longer time periods in the United States is known with some degree of precision, as data exist for a 10-year period.

Most of the data on mass concentrations of suspended particulates come from the National Air Surveillance Networks, (NASN), which uses the high-volume sampler. NASN currently operates some 200 urban and 300 nonurban stations, and is supplemented by State and local networks. From the NASN data, the annual geometric mean concentrations of suspended particulate matter in urban areas range from 60 $\mu g/m^3$ to about 200 $\mu g/m^3$. The maximum 24-hour average concentration is about three times the annual mean, but values of seven times the annual mean do occur. Mean particulate concentrations correlate, in general, with urban population class, but the range of concentrations for any class is broad, and many smaller communities have higher concentrations than larger ones. For nonurban areas the annual geometric mean is typically between 10 $\mu g/m^3$ and 60 $\mu g/m^3$.

2. Effects on Health

For the most part, the effects of particulate air pollution on health are related to injury to the surfaces of the respiratory system. Such injury may be permanent or temporary. It may be confined to the surface, or it may extend beyond, sometimes producing functional or other alterations. Particulate material in the respiratory tract may produce injury itself, or it may act in conjunction with gases, altering their sites or their modes of action.

Laboratory studies of man and other animals show clearly that the deposition, clearance, and retention of inhaled particles is a very complex process, which is only beginning to be understood. Particles cleared from the respiratory tract by transfer to the lymph, blood, or gastrointestinal tract may exert effects elsewhere. Few studies have investigated the possibility of eye injury by particles in the air; only transient eye irritation from large dust particles presently is known to be a problem.

The available data from laboratory experiments do not provide suitable quantitative relationships for establishing air quality criteria for particulates. The constancy of population exposure, the constancy of temperature and humidity, the use of young, normal, healthy animals, and the primary focus on short-term exposures in many laboratory studies make extrapolation from these studies of limited value for the general population, and singularly risky for special risk groups within the population. These studies do, however, provide valuable information on some of the bioenvironmental relationships that may be involved in the effects of particulate air pollution on health. The data they provide on synergistic effects show very clearly that information derived from single-substance exposures should be applied to ambient air situations only with great caution.

Epidemiological studies do not have the precision of laboratory studies, but they have the advantage of being carried out under ambient air conditions. In most epidemiological studies, indices of air pollution level are obtained by measuring selected pollutants, most commonly particulates and sulfur compounds. To use these same studies to establish criteria for individual pollutants is justified by the experimental data on interaction of pollutants. However, in reviewing the results of epidemiological investigations it should always be remembered that the specific pollutant under discussion is being used as an index of pollution, not as a physicochemical entity.

In epidemiologcial studies consistency of results at different times and places is important in determining the significance of observations. However, while polluted air has many similarities from place to place and from time to time, it is not identical in all communities or at all times, and complete consistency between epidemiological studies should not be expected. There are not a large number of suitable epidemiological studies available at present, but those that are available show some consistency in the levels at which effects were observed to occur.

Considerable data have been presented on a number of air pollution episodes in London and in New York City. In reviewing these data it should be remembered that British air pollution measurements are not entirely comparable with American measurements. The only published comparison indicates that the British method of measuring particulates tends to give somewhat lower readings than American methods.

Excess deaths and a considerable increase in illness have been observed in London at smoke levels above 750 $\mu g/m^3$ and in New York at a smokeshade index of 5–6 cohs. Sulfur oxides pollution levels were also high in both cases. These unusual short-term, massive exposures result in immediately apparent pathologic effects, and they represent the upper limits of the observed dose-response relationship between particulates and adverse effects on health.

Daily averages of smoke above 300 $\mu g/m^3$ to 400 $\mu g/m^3$ have been associated with acute worsening of chronic bronchitis patients in England. No comparable data are available in this country. Studies of British workmen found that increased absences due to illness occurred when smoke levels exceeded 200 $\mu g/m^3$.

Two recent British studies showed increases in selected respiratory illness in children to be associated with annual mean smoke levels above 120 $\mu g/m^3$. Additional health changes were associated with higher levels. These effects may be of substantial significance in the natural history of chronic bronchitis. Changes beginning in young children may culminate in bronchitis several decades later.

The lowest particulate levels at which health effects appear to have occurred in this country are reported in studies of Buffalo and Nashville. The Buffalo study clearly shows increased death rates from selected causes in males and females 50 to 69 years old at annual geometric means of 100 $\mu g/m^3$ and over. The study suggests that increased mortality may have been associated with residence in areas with 2-year geometric means of 80 $\mu g/m^3$ to 100 $\mu g/m^3$. The Nashville study suggests increased death rates for selected causes at levels above 1.1 cohs. Sulfur oxides pollution was also present during the periods studied. In neither study were the smoking habits of the decedents known.

Corroborating information is supplied from Fletcher's study of West London workers between the ages of 30 and 59. The data indicate that with a decrease of smoke pollution (yearly mean) from 140 $\mu g/m^3$ to 60 $\mu g/m^3$, there was an associated decrease in mean sputum volume. Fletcher noted that there may have been changes in the tar composition of cigarettes during the period studied; such a change could affect the findings. This study provides one of the rare opportunities to examine the apparent improvement in health that followed an improvement in the quality of the air.

3. Effects on Climate Near the Ground

Particles suspended in the air scatter and absorb sunlight, reducing the amount of solar energy reaching the earth, producing hazes, and reducing visibility. Suspended particulate matter plays a significant role in bringing about precipitation, and there is some evidence that rainfall in cities has increased as the cities have developed industrially.

Suspended particulate matter, in the concentrations routinely found in urban areas, considerably reduces the transmission of solar radiation to the ground, creating an increased demand for artificial light. The effect is more pronounced in the winter than in the summer, when particulate pollution loadings are higher, and sunlight must penetrate more air to reach the ground. For similar reasons the effect is also more pronounced during the workweek than on weekends, during industrial booms, and in higher latitudes. For a typical urban area in the United States, with a geometric mean annual particulate concentration of roughly 100 $\mu g/m^3$, the total sunlight, including that received directly from the sun and that reflected by the sky, is reduced five percent for every doubling of particle concentration. The reduction is most pronounced on ultraviolet radiation.

For urban areas in the middle and high latitudes, particulate air pollution may reduce direct sunlight by as much as one-third in the summer and as much as two-thirds in the winter. This effect may have implications for the delicate heat balance of the earth's atmospheric system. In spite of an increase in the carbon dioxide content of the atmosphere over the past several decades, which would presumably bring about an increase in atmospheric temperature, mean worldwide temperatures have been decreasing since the 1940's. Increased reflection of solar radiation back to outer space, brought about by increased concentrations of particulate air pollution, may be more than cancelling out the climatic effect of the increased carbon dioxide. That worldwide particulate air pollution has been increasing is evidenced by the fact that in the United States and in other countries, turbidity, a phenomenon produced by the back-scattering of direct sunlight by particles in the air, has increased significantly over the last several decades.

4. Effects on Visibility

Particles suspended in the air reduce visibility, or visual range, by scattering and absorbing light coming from both an object and its background, thereby reducing the contrast between them. Moreover, suspended particles scatter light into the line of sight, illuminating the air between, to further degrade the contrast between an object and its background.

The scattering of light into and out of the line of viewing by particles in the narrow range of 0.1 μ to 1 μ in radius has the greatest effect on visibility. Certain characteristics of behavior of these particles make it possible to formulate a useful approximate relationship between visual range and concentrations of particulate matter:

$$L_v \simeq \frac{A \times 10^3}{G'}$$

where G' = particle concentration ($\mu g/m^3$),
L_v = equivalent visual range, and
$A = 1.2^{2.4}_{0.6}$ for L_v expressed in kilometers and $0.75^{1.5}_{0.38}$
for L_v expressed in miles

The value 1.2 for A is the mid-range value empirically obtained from observations in a variety of air pollution situations. The data indicate that the range 0.6 to 2.4 covers virtually all cases studied. The relationship does not hold at relative humidities above 70 percent, nor does it apply to fresh plumes from stacks, and it may not hold for the products of photochemical reactions. A companion document, *Air Quality Criteria For Sulfur Oxides*, discusses a relationship between levels of sulfur dioxide and visual range at various relative humidities.

Within the limitations prescribed, the relationship provides a useful means of estimating approximate visual range from particulate concentrations. In addition to aesthetic degradation of the environment, reduced visibility has serious implications for safe operation of aircraft and motor vehicles. At a visual range of less than 5 miles, operations are slowed at airports because of the need to maintain larger distances between aircraft. Federal Aviation Administration restrictions on aircraft operations become increasingly severe as the visual range decreases below 5 miles. Using the upper and lower bounds of the relationship described above, visibility could be 5 miles at a particulate loading as high as 300 $\mu g/m^3$ or as low as 75 $\mu g/m^3$. However, on the average, visibility can be expected to be reduced to approximately 5 miles at a particulate concentration of 150 $\mu g/m^3$. At a level of 100 $\mu g/m^3$, visibility is reduced to $7\frac{1}{2}$ miles. This limited distance, however, may be related to particulate concentrations as low as 50 $\mu g/m^3$ and as high as 200 $\mu g/m^3$.

5. Effects on Materials

Particulate air pollution causes a wide range of damage to materials. Particulate matter may chemically attack materials through its own instrinsic corrosivity, or through the corrosivity of substances absorbed or adsorbed on it. Merely by soiling materials, and thereby causing their more frequent cleaning, particulates can accelerate deterioration.

Laboratory and field studies underscore the importance of the combination of particulate matter and corrosive gases in the deterioration of materials. On the basis of present knowledge, it is difficult to evaluate precisely the relative contribution of each of the two classes of pollution; however, some general conclusions may be drawn.

Particulates play a role in the corrosion of metals. In laboratory studies, steel test panels that were dusted with a number of active hygroscopic particles commonly found in the atmosphere corroded even in clean air. Corrosion rates were low below a relative humidity of 70 percent; they increased at relative humidities above 70 percent; and they greatly increased when traces of sulfur dioxide were added to the laboratory air.

It is apparent that the accelerated corrosion rates of various metals in urban and industrial atmospheres are largely the result of relatively higher levels of particulate pollution and sulfur oxides pollution. High humidity and temperature also play an important synergistic part in this corrosion reaction. Studies show increased corrosion rates in industrial areas where air pollution levels, including sulfur oxides and particulates, are higher. Further, corrosion rates are higher during the fall and winter seasons when particulate and sulfur oxides pollution is more severe, due to a greater consumption of fuel for heating.

Steel samples corroded 3.1 times faster in the spring of the year in New York City, where annual particulate concentrations average 176 $\mu g/m^3$, than did similar samples in State College, Pennsylvania, where the average concentrations were estimated to range from 60 $\mu g/m^3$ to 65 $\mu g/m^3$. In the fall of the year, when particulate and sulfur oxide concentrations in New York were considerably higher than in the spring, the steel samples in New York corroded six times faster than the samples at State College. Similar findings were reported for zinc samples. Moisture may have contributed to the corrosion.

In Chicago and St. Louis, steel panels were exposed at a number of sites, and measurements taken of corrosion rates and of levels of sulfur dioxide and particulates. In St. Louis, except for one exceptionally polluted site, corrosion losses correlated well with sulfur dioxide levels, averaging 30 percent to 80 percent higher than losses measured in nonurban locations. Sulfation rates in St. Louis, measured by lead peroxide candle, also correlated well with weight loss due to corrosion. Measurements of dustfall in St. Louis, however, did not correlate significantly with corrosion rates. Over a 12-month period in Chicago, the corrosion rate at the most corrosive site (mean SO_2 level of 0.12 ppm) was about 50 percent higher than at the least corrosive site (mean SO_2 level of 0.03 ppm). Although suspended particulate levels measured in Chicago with high-volume samplers also correlated with corrosion rates, a covariance analysis indicated that sulfur dioxide concentrations were the dominant influence on corrosion. Based on these data, it appears that considerable corrosion may take place (i.e., from 11 percent to 17 percent weight loss in steel panels) at annual average sulfur dioxide concentrations in the range of 0.03 ppm to 0.12 ppm, and although high particulate levels tend to accompany high sulfur dioxide levels, the sulfur dioxide concentration appears to have the more important influence.

Particulate air pollution damages electrical equipment of all kinds. Oily or tarry particles, commonly found in urban and industrial areas, contribute to the corrosion and failure of electrical contacts and connectors. Dusts can interfere with contact closure, and can abrade contact surfaces. Hygroscopic dusts will absorb water and form thin electrolytic films which are corrosive.

Particulates soil and damage buildings, statuary, and other surfaces. The effects are especially severe in urban areas where large quantities of coal and sulfur-bearing fuel oils are burned. Particles may act as reservoirs of acids, and thereby sustain a chemical attack that will deteriorate even the more resistant kinds of masonary. Particles stick to surfaces, forming a film of tarry soot and grit which oftentimes is not washed away by rain. Considerable money and effort have been spent in many cities to sandblast the sooty layers that accumulate on buildings. Water-soluble salts, commonly found in urban atmospheres, can blister paint. Other particles may settle on newly painted surfaces, causing imperfections, thereby increasing the frequency with which a surface must be painted.

The soiling of textiles by the deposition of dust and soot on fabric fibers not only makes them unattractive, and thereby diminishes their use, but results in abrasive wear of the fabric when it is cleaned. Vegetable fibers, such as cotton and linen, and synthetic nylons are particularly susceptible to chemical attack by acid components of airborne particles.

6. Economic Effects of Atmospheric Particulate Matter

It is not possible at the present stage of knowledge to provide accurate measures of all the costs imposed on society by particulate air pollution. Selected categories of effects can be quantified; it is obvious that these estimates represent a significant understatement of the total cost.

7. Effects on Vegetation

Relatively little research has been carried out on the effects of particulate air pollution on vegetation, and much of the work that has been performed has dealt with specific dusts, rather than the conglomerate mixture normally encountered in the atmosphere. This document reports briefly on some of these specific particulate studies only to illustrate the possible mechanisms through which particulate matter may affect vegetation. This information is not presented for the purpose of establishing air quality standards on these specific pollutants.

There is considerable evidence that cement-kiln dusts can damage plants. A marked reduction in the growth of poplar trees 1 mile from a cement plant was observed after cement production was more than doubled. Plugging of stomates by the dust may have prevented the exchange of gases in leaf tissue that is necessary for growth and development. Moderate damage to bean plants occurred when the plant leaves were dusted at the rate of 0.47 mg/cm^2-day (400 tons/m^2-month) for 2 days and then exposed to natural dew. The mechanism through which the leaves are damaged is not entirely understood, but direct alkaline damage to tissues beneath the crust formed by the dust and moisture has been observed. The deposits may also plug stomates and block light needed for photosynthesis. Cement-kiln dusts may change the alkalinity of soils to benefit or harm vegetation, depending on the species.

Dust deposits may also eliminate predators, and thereby bring on increased insect injury to plants; they may interefere with pollen germination; and they may make plants more susceptible to pathogens.

Fluoride dusts apparently have a difficult time penetrating leaf tissue in physiologically active form, and they are much less damaging to vegetation than is gaseous fluoride. Soluble fluoride dusts may be absorbed by the plant, but the amount is relatively small compared to that which can enter the plant in gaseous form. The evidence suggests that there is little effect on vegetation at fluoride particulate concentrations below 2 $\mu g/m^3$. Concentrations of this magnitude and above can sometimes be found in the immediate vicinity of sources of fluoride particulate pollution; they are rarely found in urban atmospheres.

Ingestion of particles deposited on plants can be harmful to animal health. Fluorosis and arsenic poisoning have been brought on through this medium.

Soot may clog stomates and may produce necrotic spotting if it carries with it a soluble toxicant, such as one with excess acidity. Magnesium oxide deposits on soils have been shown to reduce plant growth, while iron oxide deposits on soils have been shown to reduce plant growth, while iron oxide deposits appear to have no harmful effects, and may be beneficial.

8. Effects on Public Concern

Several studies indicate that there is a relationship between levels of particulate pollution, used as an index of air pollution, and levels of public concern over the problem. A study conducted in 1963 in the St. Louis metropolitan region found a direct linear relationship between the fraction of a community's population who said air pollution was a nuisance, and the annual mean concentration of particulate air pollution in the community. The relationship, which was derived from data on communities in the St. Louis area whose annual concentrations ranged from 50 $\mu g/m^3$ to 200 $\mu g/m^3$, was formulated as:

$$y = 0.3x - 14$$

where y = population fraction (%) concerned, and
x = annual geometric mean particle concentration ($\mu g/m^3$).

It is thought that the reaction to suspended particulates as a nuisance probably occurs at peak concentrations, and not necessarily at the values representing annual means. However, the relationship provides a useful example of how the nuisance effect of air pollution relates to concentrations. Approximately 10 percent of the study population considered air pollution a nuisance in areas with suspended particulates at an annual geometric mean concentration of 80 $\mu g/m^3$. At this same level of pollution, 30 percent of the study population was "aware of" air pollution. In areas with 120 $\mu g/m^3$ (annual geometric mean), 20 percent were "bothered by" and 50 percent were "aware of" air pollution; in areas with an annual geometric mean of 160 $\mu g/m^3$, one-third of the population interviewed were "bothered by" and three-fourths were "aware of" air pollution.

Although data from other studies do not readily lend themselves to quantitative formulation, they do, in general, support the relationship reported by the St. Louis study. A study of communities in the Nashville, Tennessee, metropolitan area in 1957 found that at least 10 percent of the population expressed concern about the nuisance of air pollution at dustfall levels exceeding 10 tons/mi^2-month.

9. Suspended Particles as a Sources of Odor

Particulate air pollution is not ordinarily considered a significant source of odors. However, there is evidence that liquid and even solid particles of some substances may be volatile enough to vaporize in the nasal cavity, and produce sufficient gaseous material to stimulate the sense of smell. Further, particles may carry absorbed odorants into the nasal cavity, and there transfer them to olfactory receptors. A survey of State and local air pollution control officials revealed that approximately one-fourth of the most frequently reported odors are those which are known to be, or are suspected to be, associated with particulate air pollution. The sources of these odorous particles are diverse, including diesel and gasoline engine exhausts, coffee-roasting operations, paint spraying, street paving, and the burning of trash.

B. CONCLUSIONS

The conclusions which follow are derived from a careful evaluation by the National Air Pollution Control Administration of the foreign and American studies cited in previous chapters of this document. They represent the Administration's best judgment of the effects that may occur when various levels of pollution are reached in the atmosphere. The data from which the conclusions were derived, and the qualifications which should be considered in using the data, are identified by chapter reference in each case.

1. Effects on Health

Analyses of numerous epidemiological studies clearly indicate an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain individuals with impaired health who are particularly susceptible to the adverse effects of elevated levels of particulate matter and sulfur oxides. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these changes are difficult to detect statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily demonstrated in major urban areas.

For the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can show with confidence the small changes in daily mortality which are associated with fluctuation in pollution concentrations. Such analysis has thus far been attempted only in London and in New York.

The association between longer-term community exposures to particulate matter and respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status must still be considered consequential.

Based on the above guidelines the following conclusions are listed in order of reliability, with the more reliable conclusions first. Refer to Chapter 11 for cautions to be taken in comparing British and American air quality measurement data.

a. AT CONCENTRATIONS OF *750 $\mu g/m^3$* and higher for particulates on a 24-hour average, accompanied by sulfur dioxide concentrations of 715 $\mu g/m^3$ and higher, *excess deaths* and a considerable *increase in illness* may occur. (British data; see Chapter 11, Section C–1)

b. A DECREASE FROM *140 $\mu g/m^3$ to 60 $\mu g/m^3$* (annual mean) in particulate concentrations may be accompanied by *a decrease in mean sputum volume* in industrial workers. (British data; see Chapter 11, Section C–4)

c. IF CONCENTRATIONS ABOVE *300 $\mu g/m^3$* for particulates persist on a 24-hour average and are accompanied by sulfur dioxide concentrations exceeding 630 $\mu g/m^3$ over the same average period, *chronic bronchitis* patients will likely suffer *acute worsening of symptoms.* (British data; see Chapter 11, Section C–3)

d. AT CONCENTRATIONS OVER *200 $\mu g/m^3$* for particulates on a 24-hour average, accompanied by concentrations of sulfur dioxide exceeding 250 $\mu g/m^3$ over the same average period, *increased absence of industrial workers due to illness* may occur. (British data; see Chapter 11, Section C–5)

e. WHERE CONCENTRATIONS RANGE FROM *100 $\mu g/m^3$ to 130 $\mu g/m^3$* and above for particulates (annual mean) with sulfur dioxide concentrations (annual mean) greater than 120 $\mu g/m^3$, children residing in such areas are likely to experience *increased incidence* of certain *respiratory diseases.*

f. AT CONCENTRATIONS ABOVE *100 $\mu g/m^3$* for particulates (annual geometric mean) with sulfation levels above 30 mg/cm^2-mo., *increased death rates* for persons over 50 years of age are likely. (American data; see Chapter 11, Section C–2)

g. WHERE CONCENTRATIONS RANGE FROM *80 $\mu g/m^3$ to 100 $\mu g/m^3$* for particulates (annual geometric mean) with sulfation levels of about 30 mg/cm^2-mo., *increased death rates* for persons over 50 years of age may occur. (American data; see Chapter 11, Section C–2)

2. Effects on Direct Sunlight

AT CONCENTRATIONS RANGING FROM *100 $\mu g/m^3$ to 150 $\mu g/m^3$* for particulates, where large smoke turbidity factors persist, in middle and high latitudes direct sunlight is reduced up to one-third in summer and two-thirds in winter. (American data; see Chapter 2, Section C–2)

3. Effects on Visibility

AT CONCENTRATIONS OF ABOUT *150 $\mu g/m^3$* for particulates, where the predominant particle size ranges from 0.2 μ to 1.0 μ and relative humidity is less than 70 percent, *visibility is reduced* to as low as 5 miles. (American data; see Chapter 3, Section E–4)

4. Effects on Materials

AT CONCENTRATIONS RANGING FROM 60 $\mu g/m^3$ (annual geometric mean), to *180 $\mu g/m^3$* for particulates (annual geometric mean), in the presence of sulfur dioxide and moisture, *corrosion of steel and zinc* panels occurs at an accelerated rate. (American data; see Chapter 4, Section B)

5. Effects on Public Concern

AT CONCENTRATIONS OF APPROXIMATELY *70 $\mu g/m^3$* for particulates (annual geometric mean), in the presence of other pollutants, public awareness and/or *concern for air pollution* may become evident and increase proportionately up to and above concentrations of 200 $\mu g/m^3$ for particulates. (See Chapter 7, Section B–1)

C. RESUME

In addition to health considerations, the economic and aesthetic benefits to be obtained from low ambient concentrations of particulate matter as related to visibility, soiling, corrosion, and other effects should be considered by organizations responsible for promulgating ambient air quality standards. Under the conditions prevailing in areas where the studies were conducted, adverse health effects were noted when the annual geometric mean level of particulate matter exceeded 80 $\mu g/m^3$. Visibility reduction to about 5 miles was observed at 150 $\mu g/m^3$, and adverse effects on materials were observed at an annual mean exceeding 60 $\mu g/m^3$. It is reasonable and prudent to conclude that, when promulgating ambient air quality standards, consideration should be given to requirements for margins of safety which take into account long-term effects on health and materials occurring below the above levels.

APPENDICES

APPENDIX A—SYMBOLS

A	area of the filter stain, cm^2
B	turbidity coefficient (empirical), $\lambda = 0.5\mu$
C	particle concentration in the air, $\mu g/m^3$
B_o	turbidity coefficient, measured on the ground
B_z	turbidity coefficient, measured at height z above the ground
I	light intensity, usually in $ergs/cm^2/sec$
I_o	initial or incident light intensity
L_v	equivalent visual range, or visibility, but calculated on basis of a constant scattering coefficient, b, along the line of sight
P	probability
$P(\lambda)$	solar transmission factor for atmosphere aerosols; it is a function of λ
Q	particle mass concentration in the air, usually in $\mu g/m^3$
S	scattering cross-section, cm^2/particle
S_c	derived surface particle concentration, $\mu g/cm^2$
T	transmissivity, I/I_o
V	volume of air sampled, m^3
b	extinction coefficient, m^{-1} $(= b_{\text{abs aerosol}} + b_{\text{abs gas}} + b_{\text{Rayleigh}} + b_{\text{scat}})$
$b_{\text{abs aerosol}}$	extinction coefficient due to absorption by aerosol particles
$b_{\text{abs gas}}$	extinction coefficient due to absorption by gas such as NO_2
b_{Rayleigh}	extinction coefficient due to scatter by gas molecules
b_{scat}	extinction coefficient due to scatter by aerosols
d	diameter of a spherical particle, cm
e	Napierian log base (=2.7182818)
g	acceleration of gravity, cm/sec^2
m	index of refraction
γ	correlation coefficient
v	settling velocity of a particle, cm/sec
x	distance, m or cm
z	height above ground, meters
α	light scattering coefficient (empirical) relates b to λ
β	turbidity coefficient for $\lambda = 1\mu$
v	viscosity of air, or other gas, poises
λ	wavelength of light Ångstroms, microns, or nanometers
x	probability function, usually expressed as x^2 ("Chi square")

APPENDIX B—ABBREVIATIONS

Å	Ångstrom, $1\text{Å} = 10^{-8}$ cm	m^3	cubic meter
BTPS	body temperature, pressure, saturated	mg	milligram
		mi	mile
cm	centimeter, $1\text{cm} = 10^{-2}\text{m}$	mi^2	square mile
cm^2	square centimeter	min	minute
cm^3	cubic centimeter	ml	milliliter
CMD	count median diameter	MMD	mass median diameter
coh	coefficient of haze	mo	month
DMBA	10-dimethyl-1, 2 benzanthracene	mu	millimicron, $1\text{m}\mu = 0.001\mu$
FEV	forced expiratory volume	μ	micron, $1\mu = 10^{-4}\text{cm} = 10^{-6}\text{m} = 10^4\text{Å}$
FVC	forced vital capacity	μg	microgram
g	mass, grams	ppm	parts per million
hr	hour	RUDS	reflectance unit of dirt shade
l	liter	sec	second
m	length, meter	yr	year

APPENDIX C—CONVERSION FACTORS

To convert—	*To—*	*Multiply by—*
mg/m^3	μg/m^3	1000
mg/100 m^3	μg/m^3	10
μg/ft^3	μg/m^3	35.314
μg/1000 m^3	μg/m^3	10^{-4}
μg SO_2/m^3 (0°C, 760 mm Hg)	ppm SO_2 (vol)	3.5×10^{-4}
ppm SO_2 (vol)	SO_2 μg/m^3 (0° C, 760 mm Hg)	2860
tons/mi^2-mo	mg/cm^2-mo	3.5×10^{-2}
tons/mi^2-mo	g/m^2-day	1.07×10^{-2}
tons/mi^2-mo	metric tons/km^2-mo	3.5×10^{-1}
tons/mi^2-mo	μg/m^2-mo	3.5×10^{5}
mg/cm^2-mo	tons/mi^2-mo	28.5
g/m^2-day	tons/mi^2-mo	85.5
μ or μm	m	10^{-6}
μ	Å	10^{4}
Å	cm	10^{-8}
μ	cm	10^{-4}
bbl	gal.	55
1	ft^3	0.0353
ft^3	1	28.32
ml	in^3	6.1×10^{-2}
m	ft	3.28
lb	g	453.6

APPENDIX D—GLOSSARY

Acid, free—an acid which is unneutralized by other compounds

Acid, stearic—the most common fatty acid occurring in natural animal and vegetable fats, almost completely colorless and odorless

Acrolein—a toxic colorless mobile liquid aldehyde with an acrid odor

Adenocarcinoma—a malignant tumor in which the cells are arranged in the form of glands or gland-like structures

Adenoma—an epithelial tumor, usually benign, with a gland-like structure

Adhesion—the attraction of two unlike substances

Adsorption—the phenomenon by which gases are attracted, concentrated, and retained at a boundary surface

Aerodynamics—a phase of the mechanics of fluids, its study being limited to the reactions caused by relative motion between fluid and solid, the fluid being limited to air in most cases but occasionally broadened to include any gas

Aerosol—a cloud of solid particles and/or liquid droplets smaller than 100μ in diameter, suspended in a gas

Aerosol, bidisperse—an aerosol in which all the suspended particles tend to be of two sizes (diameter)

Aerosol, monodisperse—an aerosol in which all the suspended particles are of nearly equal size (diameter)

Air, residual—the air that stays in the lungs after forceful expiration

Air, tidal—the air that is carried to and from the lungs during a respiratory cycle

Airway—any part of the respiratory tract through which air passes during breathing

Airway resistance—resistance to the flow of air in the passages to the lungs

Albedo—the ratio of the amount of electromagnetic radiation reflected by a body to the amount incident upon it, commonly expressed as a percentage

Aldehyde—any of a class of organic compounds containing the group R–CHO, intermediate in state of oxidation between primary alcohols and carboxylic acids

Alveolus (pl. *alveoli*)—a small, sac-like dilation at the innermost end of the airway, through whose walls gaseous exchange takes place

Analysis, factorial—a method of evaluating certain definite integrals by the use of gamma functions

Anaplasia (adj. *anaplastic*)—a condition in tumor cells in which normal functional and physical differentiation is lost

Anthracosis—a disease of the lungs caused by inhalation and accumulation of carbon particles

Antirachitic—opposing or preventing the development of rickets

Aphid (aphis)—a small sucking insect; a plant louse

Atelectasis—the collapse of all or part of a lung, with resultant loss of functioning tissue

Attenuation—In physics, any process in which the flux density (or power, amplitude, intensity, illuminance, etc.) of a "parallel beam" of energy decreases with increasing distance from the energy source

Auscultation (adj. *Auscultatory*)—the act of listening for sounds within the body, usually with the use of a stethoscope

Bacillus subtilis—a common microorganism found in soil and water and frequently occurring as a laboratory contaminant; rarely implicated in causing disease

Benzo (a) pyrene—a polycyclic aromatic hydrocarbon which under certain circumstances has been shown to produce cancer

Bifurcation—a site where a single structure divides into two branches

Blistering, osmotic—paint blisters caused by moisture picking up water-soluble salts in its passage through the paint film, resulting in an ideal situation for osmosis which represents a tremendous force formation of blisters

Blowby—the leakage of gas or liquid between a piston and its cylinder during operation

Body, asbestos—a fiber of asbestos surrounded by a deposit of protein material

Bronchiectasis—a chronic dilatation of a bronchial passage

Bronchiole—one of the finer subdivisions of the bronchial tree

Bronchitis—an inflammation of the bronchi, usually manifest clinically by cough and the production of sputum

Bronchitis, chronic—a long-standing inflammation of the bronchi characterized by excessive mucus secretion in the bronchial tree and manifested by a persistent or recurrent productive cough. For the purposes of definition, these symptoms must be present on *most days* for a minimum of *3 months* of the year for at least *2* successive *years* (American Thoracic Society)

Bronchoconstriction—a diminution in the size of the lumen of a bronchus

Bronchus (pl. *bronchi*)—one of the larger air passages in the lung

Broth—a liquid medium for the cultivation of microorganisms

Capacity, forced vital (FVC)—the largest amount of gas which can be forcibly expired from the lungs following a maximal inspiration

Capacity, functional residual (FRC)—the volume of gas remaining in the lungs at the resting end-expiratory level

Capacity, total lung (TLC)—the volume of gas contained in the lungs at full inspiration

Capacity, vital—the maximum volume of gas which can be expired from the lungs following a maximum inspiration

Carbonation—conversion into a carbonate (which in many cases refers to one or more members of the calcite, dolomite, and aragonite groups of minerals) impregnation with carbon dioxide

Carcinogen—a substance capable of causing living tissue to become cancerous

Carcinogenesis—the production of cancer

Carcinoma—cancer; malignant growth made up of cells derived from epithelial tissue

Carcinoma, bronchogenic—cancer arising in bronchial tissue of the lung

Carcinoma, squamous cells—cancer developing from squamous epithelial cells

Carcinoma in situ—a neoplastic entity in which tumor cells are present but the invasion of normal tissue has not yet taken place

Cardoivascular—pertaining to the heart and blood vessels

Cercospora beticola—a member of the genus *Cercospora*, which consists of imperfect fungi that are leaf parasites with long slender multi-septate spores

Channel black—carbon black made by impingement of a luminous natural gas flame against an iron plate from which it is scraped at frequent intervals; properties vary widely, but the material has an unusually fine state of subdivision and great surface area

Chloroplast—a specialized body (a plastid) containing chlorophyll in the cytoplasm of plants; the site of photosynthesis and starch formation in plants

Cholinesterase—any one of several enzymes that hydrolyze choline esters, occurring most frequently in nervous tissue and the blood

Cilium (pl. *cilia*)—small, hairlike process attached to a free surface of a cell, capable of rhythmic movement

Clearance—the removal of material from the body or from an organ

Clinker—kiln-fired limestone from which commercial cement is made

Coalescence—in cloud physics, the merging of two water drops into a single larger drop

Coefficient, absorption—the fractional rate at which flux density of radiation decreases by absorption with respect to the thickness of the absorbing medium traversed

Coefficient, extinction—the sum of the absorption coefficient and the scattering coefficient for a medium that both absorbs and scatters radiation

Coefficient, scattering—the fractional rate in the transmission of radiation through a scattering medium (as of light through fog) at which the flux density of radiation decreases by scattering in respect to the thickness of the medium traversed

Coh unit—a measure of light absorption by particles, defined as that quantity of light scattering solids producing an optical density of 0.1

Collagen—a fibrous protein forming the main supportive structure of connective tissue

Collector, cyclonic—a centrifugal fractionator in which a vortex of air throws particles out of a stream, where they collect or stick to the surface of a container

Comminute—to break or crush into small pieces

Concentration—the total mass (usually in micrograms) of the suspended particles contained in a unit volume (usually one cubic meter) at a given temperature and pressure; sometimes, the concentration may be expressed in terms of total *number* of particles in a unit volume (e.g., parts per million); concentration may also be called the "loading" or the "level" of a substance concentration may also pertain to the strength of a solution

Conifer—belonging to the coniferales order, consisting primarily of evergreen trees and shrubs

Consolidation—the process by which a diseased lung passes from an aerated collapsible state to one of an airless solid consistency because of accummulation of exudate

Contrast—in visual range theory, the ratio of the apparent luminance of a target minus that of its background to the apparent luminance of the background

Conurbation—a great aggregation or continuous network of urban communities

Coryza—an acute catarrhal condition of the nasal mucous membrane with profuse discharge from the nostrils

Criteria, air quality—a compilation of the scientific knowledge of the relationship between various concentrations of pollutants in the air and their adverse effects

Cryolite—a mineral fluoride consisting also of sodium and aluminum

Cuticle—a varnish-like layer covering the surface of a leaf

Cytoplasm—the protoplasm of a cell (excluding that of the nucleus)

Dead space, anatomic—that part of the airway occupied by gas which is unavailable (by its location) to take part in oxygen-carbon dioxide exchange through the walls of the alveoli

Dead space, physiologic—the volume of gas within the alveoli which does not participate in the oxygen-carbon dioxide exchange through the walls of the alveoli

Dehydrogenase—any one of various enzymes which accelerate the removal of hydrogen from metabolities and its transfer to other substances, thus playing an important role in biological oxidation-reduction processes

Deliquesce—to dissolve gradually and become liquid by absorbing moisture from the air

Density—the amount of matter per unit volume, usually expressed in grams per cubic centimeter

Density, optical—the degree of opacity of any translucent medium; the common logarithm of the ratio of the initial intensity of light to the intensity of transmitted or reflected light

Desorption—the release of a substance which has been taken into another substance by a physical process or held in concentrated form upon the surface of another substance; the reverse of absorption or adsorption

Desquamate—to cast off epidermis in shreds or scales; to peel off in sheets or scales

Deviation, standard geometric (σg)—a measure of dispersion of values about a geometric mean; the portion of the frequency distribution that is one standard geometric deviation to either side of the geometric mean accounts for 68% of the total samples

Deviation, standard normal—a measure of dispersion of values about a mean value; the square root of the average of the squares of the individual deviations from the mean

Dextran—a water-soluble polymer used therapeutically as a plasma substitute

Diameter, count median (CMD)—the geometric median size of a distribution of particles, based on a numerical count

Diameter, mass median (MMD)—the geometric median size of a distribution of particles, based upon a weight (usually derived from a Stokes' Diameter)

Diameter, Stokes'—the diameter that a unit density particle of spherical shape would have if it behaved the same as the particle being studied

Dichotomous—dividing in succession into pairs; showing a dual arrangement

Distal—furthest or most remote from the median line of the body, from the point of attachment, or from the origin; peripheral (*cf.* proximal)

Diverticulum (pl. *diverticula*)—a pouch or cul-de-sac of a hollow organ

Dosimetry—the accurate measurement and determination of (medicinal) doses

Dyspnea—difficult or labored breathing

Earth, diatomaceous—a chalky material used as a filter aid, an absorbent, a filler, an abrasive, and as thermal insulation

Edema—a condition due to the presence of abnormally large amounts of fluid in the intercellular tissue spaces of the body

Effluent—something that flows out, such as a liquid discharged as a waste

Elution—the process of washing out, or removing with the use of a solvent

Elutriator, fractional—a fractional sampler which removes coarse particles from the air by gravity settlement

Emphysema—a swelling due to the presence of air, usually excess or additional air. The term is usually used to refer to pulmonary emphysema

Emphysema, pulmonary—a condition in which there is overdistension of air spaces and resultant destruction of alveoli and loss of functioning lung tissue

Endocytosis—a condition or disease arising from the inclusion within a cell of material which does not properly belong there

Entomology—zoology dealing only with insects

Epidemiology—a science dealing with the factors involved in the distribution and frequency of a disease process in a population

Epidermis—the outermost layer of skin in animals; any integument

Epiglottis—a plate of cartilage which covers the entrance to the larynx during swallowing, thus preventing food or fluid from entering the windpipe

Epithelium—a closely packed sheet of cells arranged in one or more layers, covering the surface of the body and lining hollow organs

Epithelium, columnar—a type of epithelium composed of tall, prismlike cells

Epithelium, spuamous—a type of epithelium composed of plate-like cells

Ergometer—an instrument which measures work done, e.g., by muscle contraction; a dynamometer

Evaginate—to turn inside out or protrude by eversion

Extrapolate—to project data into an area not known or experienced, and arrive at knowledge based on inferences of continuity of the data

Fibrosis—the development of fibros tissue; sclerosis

Filiform—having the shape of a thread or filament

Floc—something occurring in indefinite masses or aggregates

Fluorosis—a pathologic condition resulting from excessive intake of fluorine

Fluorspar—the mineral fluorite

Flux—a flowing or discharge of fluid; a substance used to promote fusion (as by removing impurities) of metals or minerals; the rate of transfer of fluid, particles, or energy (as radiant energy) across a given surface

Fume—an aerosol formed by the condensation of vapors as they cool

Gastric—pertaining to the stomach

Gastrointestinal—pertaining to the stomach and intestines

Goblet cell—a type of epithelial cell containing mucus and having the shape of a flask or goblet

Gravimetric—of or relating to measurement by weight

Haze—fine dust or salt particles dispersed through a portion of the atmosphere; the particles are so small that they cannot be felt or individually seen with the naked eye, but they diminish horizontal visibility and give the atmosphere a characteristic opalescent appearance that subdues all colors

Hematite—the mineral iron oxide; Fe_2O_3

Histamine—a substance which produces dilatation of capillaries and stimulates gastric secretion, occurring in both animal and vegetable tissues; β-imidazolethylamine

Histogram—a graphical representation using a series of bars

Histology—the study of the anatomy of tissues and their microscopic cellular structure

Hydrocarbon—a compound containing only hydrogen and carbon. This group is subdivided into alicyclic, aliphatic, and aromatic hydrocarbons according to the arrangement of the atoms and the chemical properties of the compounds.

Hygroscopic—readily absorbing and retaining moisture from the atmosphere

Hyperplasia (adj. *hyperplastic*)—an increase in the number of cells in and bulk of a tissue, with retention in normal function and cellular structure

Illumination—the process in which light is brought to some surface or object

Impactor, cascade—an instrument which employs several impactions in series to collect successively smaller sizes of particles

Incidence—the rate at which a certain event or disease occurs

Insolation—the rate at which direct solar radiation (of all wavelengths) is delivered to a unit area of a horizontal surface, usually at or near ground level

Insulation—the prevention of the transfer of energy between two conductors by separation of the conductors with a non-conducting material; or, the non-conducting material itself

Interstitial—pertaining to or situated in the space between cells

In vitro—in a test tube or other artificial environment

In vivo—within a living body

Isotherm—a line on a chart representing changes of volume or pressure under conditions of constant temperature; or lines on a map connecting points having the same temperature

Ketone—any of a class of organic compounds that are characterized by a carbonyl group attached to two carbon atoms, usually contained in hydrocarbon radicals or in a single bivalent radical, similar to aldehydes but less reactive

Lacrimation (*lachrymation*)—tear formation, especially in excess

Langley—a unit of energy per unit area, commonly employed in radiation theory and equal to one gram-calorie per square centimeter

Larynx—the organ concerned with the production of the voice, situated at the upper end of the trachea

Lesion—an injury or other circumscribed pathologic change in a tissue

Logarithm—a number which represents the power to which a given number must be raised to produce another given number

Lumen—the inner space of a hollow organ or tube

Lycopodium—a genus of club-moss; a powder ("vegetable sulfur") used to prevent the agglutination of pills in a box or as a dusting powder

Lymph—a fluid that is collected from the tissues throughout the body, flows in the lymphatic vessels, and is eventually added to the bloodstream

Lymphoma—any neoplasm developing from lymphatic tissue

Macrophage—a large phagocytic cell found in the connective tissue, especially in areas of inflammation

Malignancy—something which tends to become progressively worse and if unchecked could result in death

Mastoiditis—an inflammation of the skull bone behind the ear

Mean, geometric (M_g)—a measure of central tendency for a log-normal distribution;

the value, in a given set of samples, above which 50% of the values lie

Meatus—a natural body passage, particularly the external opening of a canal

Mesothelioma—a tumor which develops from the lining of a coelomic body cavity

Metaplasia (adj. *metaplastic*)—a change in the cells of a tissue to a form which is not normal for that tissue

Metastable—marked only by a slight margin of stability

Meteorological range (*standard visibility, standard visual range*)—an empirically consistent measure of the visual range of a target; a concept developed to eliminate from consideration the threshold contrast and adaptation luminance, both of which vary from observer to observer

Morbidity—the occurrence of a disease state

Morphology—a branch of biology dealing with the structure and form of living organisms

Mortality—the ratio of the total number of deaths to the total population, or the ratio of the number of deaths from a given disease to the total number of people having that disease

Motion, Brownian—the rapid random motion of small particles due to bombardment by surrounding molecules which are in thermal motion

Mucin—a glycoprotein or mucopolysaccharide secreted by mucous glandular cells

Mucopurulent—pertaining to an exudate (or sputum) that is chiefly purulent (pus) but also contains significant amounts of mucus

Mucoviscidosis—cystic fibrosis

Mucus (adj. *mucous*)—the clear viscid secretion of a mucous membrane

Mural—pertaining to the wall of a cavity

Mustard gas—an irritating and toxic volatile liquid used as a weapon in World War I; dichloroethyl sulfide

Naris (pl. *nares*)—a nostril or other opening into the nasal cavity

Nasopharynx—the part of the pharynx (throat) lying above the level of the soft palate

Nebulize—to reduce to a fine spray

Necrosis—localized death of cells

Neoplasm—any abnormal growth, such as a tumor

Nephelometer—a photometric instrument for the determination of the amount of light transmitted or scattered by a suspension of particles

Node—a circumscribed swelling

Node, lymph—one of many accumulations of lymphatic tissue situated throughout the body

Nomogram—a graph that enables one to read off the value of a dependent variable with the use of a straightedge, when the values of two or more independent variables are known

Nucleation—the process of particle growth through collection around a nucleus

Nucleus (*condensation nucleus*)—a particle in the size range from 0.1μ to 1μ which serves as a nidus on which water or other vapors in the air can condense to form liquid droplets

Olefin—a class of unsaturated aliphatic hydrocarbons of the general formula C_nH_{2n}

Olfactory—pertaining to the sense of smell

Ophthalmic—pertaining to the eye

Otitis—an inflammation of the ear

Palisade tissue—a layer of columnar cells rich in chloroplasts found beneath the upper epidermis of foliage leaves

Parameter—an arbitrary constant which characterizes a mathematical expression

Parenchyma—the specific or functional tissue of a gland or organ, as opposed to its supporting framework

Particle—any dispersed matter, solid or liquid, in which the individual aggregates are larger than single small molecules (about 0.0002μ in diameter), but smaller than about 500μ in diameter

Particulate—existing in the form of minute separate particles

Pathogenesis—the production or the mode of origin and development of a disease condition

Pathology—the study of the essential nature of disease, particularly with respect to the structural and functional changes in organs and tissues

Peritoneum—the membrane lining the abdominal cavity and investing the viscera

Phagocyte—a cell that has the power of ingesting microorganisms and other small particles

Phagocytosis—the ingestion of a microorganism or other small particle by a phagocyte

Pharynx—the upper expanded portion of the alimentary canal lying between the mouth, the nasal cavities, and the beginning of the esophagus; the throat

Photometer—an instrument for measuring luminous intensity, luminous flux, illumination, or brightness by comparison of two unequal lights from different sources

Photomicrograph—a photograph of a magnified image of a small object

Photosynthesis—the formation of carbohydrate from carbon dioxide and water in the presence of chlorophyll and light, in plant tissues

Physiology—a science which studies the function of a living organism or its parts

Phytotoxic—harmful to plant materials

Plasmolysis—the shrinking of the cytoplasm away from the wall of a living cell due to water loss by osmotic action

Plethysmograph—an apparatus for the determination and recording of a change in the size of an organ or limb or body

Pneumococcus (pl. *pneumococci*)—a bacterial organism which most often infects the lung and is a common cause of pneumonia; *Diplococcus pneumoniae*

Pneumoconiosis—a fibrous reaction in the lungs, caused by the retention of certain inhaled dusts in the lungs

Pneumonitis—a general term for inflammation of the lung

Pneumotachygraph—an instrument used to determine the force and velocity of respired air

Polystyrene—a clear, colorless polymer of styrene, an unsaturated hydrocarbon of the form $C_6H_5CH=CH_2$

Potentiation—synergism, as between two agents which together have a greater effect than the sum of their effects when acting separately

Precipitation—any or all of the forms of water particles, whether liquid or solid, that fall from the atmosphere and reach the ground. It is a major class of hydrometeor, but is distinguished from cloud, fog, dew, rime, frost, etc. in that it must "fall," and is also distinguished from cloud and virga in that it must reach the ground

Precipitator, electrostatic—an apparatus for the removal of suspended particles from a gas by charging the particles and precipitating them by applying a strong electric field

Predator—an organism living by preying on other organisms

Prevalence—the number of cases of a disease at a given time

Prodrome—the symptoms preceding the appearance or recognition of an actual disease state

Proteinosis—the accumulation of protein in excess in the tissues

Proteinosis, alveolar—a chronic progressive lung disease characterized by the accumulation of granular proteinaceous material in the alveoli

Proximal—nearest to the center of the body or the point of origin (*cf.* distal)

Radioautograph (*autoradiogram*)—a radiographic portrayal of an object or organism made by the inherent radioactivity of the object or organism

Rale—an abnormal respiratory sound heard in auscultation of the chest

Ratio, standardized mortality—the ratio of the number of deaths observed in a given population over a given period of time *to* the number of deaths expected to occur in the given population over the same period of time if the given population behaved as any other group of similar composition would during that same period

Reflectometer—a photometric or electronic device for measuring the reflectances of light or other radiant energy

Regression—a trend or shift toward a mean. A *regression curve* or *line* is thus one that best fits a particular set of data according to some principle

Rhinitis—an inflammation of the nasal mucous membrane

Rhinorrhea—"runny nose"

Rhonchus (pl. *rhonchi*)—a dry, coarse sound usually originating from partial obstruction in a bronchial tube

Scattering, Mie—any scattering produced by spherical particles, without specific regard to the comparative size of radiation wavelength or particle diameter

Scattering, Rayleigh—any scattering process produced by spherical particles whose radii are smaller than about one-tenth the wavelength of the scattered radiation

Sigmoid curve—a "bell-shaped" curve serving as a prototype for the normal distribution of data about a mean

Silicosis—a type of pneumoconiosis caused by inhalation of silica dust and characterized by silica-containing nodules of scar tissue in the lung parenchyma

Sorption—the generalized term for the many phenomena commonly included under the terms adsorption and absorption, when the nature of the phenomenon is unknown or indefinite

Spirometer—an instrument for the measurement of the volume of gas respired by the lungs

Spore—a reproductive element of many lower organisms

Squamous—resembling or covered with scales

Standards, air quality—levels of air pollutants which cannot legally be exceeded during a specific time in a specific geographical area

Stasis—the slowing down or cessation of the normal flow

Stigma—the part of the pistil of a flower which receives the pollen granules and on which they germinate

Stokes' Law—a law in physics stating that the force required to move a sphere through a given viscous fluid at a low uniform velocity is directly proportional to the velocity and radius of the sphere

Stoma (pl. *stomata*)—a small opening in the epidermis of a plant

Subcutaneous—beneath the skin

Supercool—to cool below the freezing point without solidification or crystallization

Supersaturation—a condition of containing an excess of some material or force, over the amount required for saturation normally

Synergism—a situation in which the combined action of two or more agents acting together is greater than the sum of the action of these agents separately

Systemic—relating to the body as a whole, rather than to its individual parts

Tannin—any one of a group of soluble astringent complex phenolic substances that are widely distributed in plants

Terpene—any one of a class of isomeric hydrocarbons of the prototype $C_{10}H_{16}$ that are found in many essential oils, but especially from conifers; may also refer to any of various compounds derived from terpene hydrocarbons or closely related to them

Tipburn—a disease of the potato, lettuce, and other cultivated plants, characterized by burning or browning of the tips and margins of the leaflets and caused by loss of water due to excessive heat and sunshine

Toxicology—the study of poisons, including their preparation, identification, physiologic action, and antidotes

Trachea—windpipe; the airway extending from the larynx to the origin of the two mainstem bronchi

Tracheobronchitis—an inflammation of the trachea and bronchi

Turbidity—in meteorology, any condition in the atmosphere which reduces its transparency to radiation, especially to visible radiation

Tyndallometer—an instrument that measures suspended particle concentration by the amount of light scattered out of a beam

Ultrafilterable—capable of being separated by a dense filter which is used for the filtration of a colloidal solution holding back the dispersed particles but not the liquid

Updraft—an upward movement of air or other gas

Ventilation, minute—the total volume of gas respired in one minute, i.e., the tidal volume multiplied by breaths per minute

Visibility—In United States weather observing practice, the greatest distance in a given direction at which it is just possible to see and identify with the unaided eye (a) in the daytime, a prominent dark object against the sky at the horizon, and (b) at night, a known, preferably unfocused, moderately intense light source. After visibilities have been determined around the entire horizon circle, they are resolved into a single value of prevailing visibility for reporting purposes.

Visual range—the distance, under daylight conditions, at which the apparent contrast between the specified type of target and its background becomes just equal to the threshold contrast of an observer; to be distinguished from the night visual range. The visual range is a function of the atmospheric extinction coefficient, the albedo and visual angle of the target, and the observer's threshold contrast at the moment of observation.

Volume, forced expiratory (FEV)—the volume of gas forcibly exhaled over a given time interval (usually measured in seconds) after maximum inspiration, e.g., $FEV_{1.0}$ for this measurement over a 1.0 second period.

Volume, minute—same as minute ventilation

Volume, tidal—the volume of gas inspired or expired during each respiratory cycle.

210

AUTHOR INDEX

Ackley, C., 94
Agnese, G., 162, 172–173
Ahlquist, N. C., 56, 57, 59
Albert, R. E., 122
Alcocer, A. E., 26
Altshuler, B., 113, 117, 118
Amberg, H. R., 106
Amdur, M. O., 113, 131, 132, 133, 134, 136
Andersen, A. A., 17, 19
Anderson, D. O., 150, 161, 164, 165, 173–174
Anderson, P. J., 92
Andrea, J., 140
Angel, J. H., 168, 175
Ångstrom, A. K., 38, 39, 43, 53
Antler, M., 69
Antweiler, H., 121
Arnett, L. C., 122
Ashworth, J. R., 41, 44
Auerbach, O., 141
Axt, C. J., 19
Ayer, H. E., 137

Baetjer, A. M., 132
Balzer, J. L., 137
Barfinkel, L., 141
Barlett, F. E., 106
Bates, D. V., 163, 173–174
Baulch, D. M., 19, 37
Baumberger, J. P., 117
Bechmann, H., 117
Becker, W. H., 165, 173–174
Belton, J., 154
Berge, H., 94
Biersteker, K., 168, 175
Black, S., 118
Blifford, I. H., 11
Bohne, H., 91, 92, 94
Bonser, G. M., 139
Boren, H. C., 135
Bowden, A. T., 26
Bownes, K., 105
Bradley, W., 152, 154, 156, 171, 172
Brandt, P., 17
Brasser, L. J., 150, 153, 158
Braverman, M. M., 153, 154, 171
Brieger, H., 122
Brier, G. W., 41
Briscoe, W., 122
Brooke, A. G. F., 71
Brown, C. E., 117
Brown, D. A., 157, 172
Brown, J. H., 118
Brown, M. C., 137
Bryson, R. A., 42, 43
Buck, S. F., 157, 172
Bunyard, F. L., 99
Burgess, F., 130, 131, 136
Burgess, S. E., 151, 171
Burn, J. L., 157, 158, 159, 164, 172–173, 174
Bye, W. E., 167

Cadle, R. D., 8, 10
Cambell, J. A., 137
Capps, R., 135
Carey, W. F., 69
Carnes, W. H., 141
Cartwright, J., 69, 71
Casarett, L. J., 122, 123
Cassel, E. G., 150, 154, 156, 171, 172
Cefis, F., 139
Chaney, A. L., 26
Changnon, S. A., Jr., 41
Charlson, R. J., 19, 54, 55, 56, 57, 59
Chass, R. L., 24
Christofano, E. E., 135, 136
Clark, J. G., 22
Clark, W. E., 23, 54
Clemo, G. R., 137
Coblentz, H., 39
Coffin, D. L., 129
Collet, A., 123
Collins, G. F., 106
Conner, W. D., 53
Cook, K. M., 118
Cooley, R. N., 132
Cooper, W. C., 137
Copson, H. R., 66, 67, 68, 69
Corn, M., 6, 8, 19, 132, 133
Coughanower, D. R., 136
Cralley, L. J., 137
Creasia, D. A., 133
Crider, W. L., 26
Crowley, D., 154, 155
Cuffe, S. T., 5
Cullumbine, H., 130, 132, 136
Czaja, A. T., 91, 92

Dalhamn, T., 121, 122, 135
Darley, E. F., 90, 91, 92, 93
Dautrebande, L., 117, 119, 133, 135
Davies, C. N., 111, 117, 118, 119, 121, 122, 123, 132, 135, 136
Davies, R. I., 157, 171–172
Day, J. A., 11
De Groot, I., 99, 101
Delly, J. G., 18
De Maio, L., 19
Dennis, W. L., 118
Dessens, J., 18
De Treville, R. T., 135
Devir, S. E., 131
Diamond, J. R., 149, 155, 156, 171, 172
Dickerson, R. C., 20, 22
Diem, M., 17
Djordjevic, N., 11
Dobrogorski, O. J., 130
Dohan, F. C., 164
Doherty, R. E., 26
Douglas, J. W. B., 165, 173–174
Downs, W. L., 122
Draftz, R. G., 18
Draper, P., 26
Drinker, P., 8, 113, 117, 118, 132
Drolette, B. M., 153, 154, 171
Du Bois, A. D., 133

Eckel, O., 39
Edmonds, S. M., 106
Elliott, A., 166
Ellis, O. B., 66, 67, 68
Ellison, J. M., 22
Epstein, S. S., 140
Erdhardt, C., 153, 154, 171

Falk, H. L., 120, 134, 137, 138, 141
Faoro, R. B., 16
Faulds, J. S., 139
Fensterstock, J. C., 16
Ferris, B. G., Jr., 164, 173–174
Field, F., 153, 154, 171
Fieldner, A. C., 117
Findeisen, W., 114, 115, 119
Finn, J. L., 117
Firket, J., 150
Fischer, W. H., 19
Fish, B. R., 10
Fisher, M., 121
Flesch, J., 132
Fletcher, C. M., 168, 175
Fochtman, E. C., 71
Foster, K. E., 17
Frank, E. R., 20, 23
Frederick, R. H., 41
Friedlander, S. K., 54, 58

Gates, D. M., 38
Georgii, H. W., 41
Gibb, F. R., 117, 119, 121
Gilbert, J., 69
Giles, C. H., 69
Giner, R., 40
Girorer, W., 117
Glasser, M., 154, 155
Goetz, A., 6, 10, 19, 58, 59, 106
Gong, W. K., 26
Gore, A. T., 151, 171
Graf, P., 132
Graff-Baker, C., 71
Greenburg, L., 68, 69, 153, 154, 171
Greenwood, D. A., 95
Greenwood, J. A., 69
Greezuitay, L. A., 139
Gross, P., 111, 123, 135
Grote, M. J., 139
Gruber, C. W., 17
Gucker, F. T., 19
Guderian, R., 92

Haddow, A., 139
Hagstrom, R. M., 159, 161, 172–173
Haines, G. F., Jr., 20
Hamilton, R. J., 17
Hammond, C., 130
Hammond, E. C., 141
Hand, I. F., 40
Handyside, A. J., 166, 168, 174–175
Harris, J., 69
Harris, W. B., 17
Harrison, L. E., 95
Hatch, T. F., 8, 111, 119, 120, 123
Haythorn, S. R., 131
Helwig, H. L., 26
Hemeon, W. C. L., 17, 20
Hendrix, W. G., 23
Hess, V. F., 40
Hewson, E. W., 17
Hilding, A. C., 122
Hill, A. C., 93, 94
Hill, I. D., 148, 168, 175
Hitchcock, A. E., 93, 94
Hodkinson, J. R., 53
Hoffman, P., 19
Holbrow, G. L., 71
Holden, F. R., 95
Holland, W. W., 162, 163, 166, 167, 172–173, 174–175
Holma, B., 122
Holzworth, G. C., 51
Horning, E. S., 139
Horton, R. J. M., 164
Horvath, H., 19, 54, 56, 57, 59
Hudson, J. D., 66, 67
Hueper, W. C., 137
Huey, N., 81
Humphreys, W. J., 43
Hurd, F. K., 23

Ide, H. M., 20
Imada, M., 57

Jacobs, M. B., 65, 69, 153, 154, 171
Jacobson, J. S., 93, 94
Jennings, O. E., 94
Jens, W., 24, 25
Johnstone, H. F., 136
Jones, E. E., 26
Joosting, P. E., 151, 153, 158
Joshi, S., 140
Junge, C. E., 11, 23, 53, 54
Jurksch, G., 17
Jutze, G. A., 17

Kaiser, E. R., 25, 107
Kakis, F., 105
Kallai, T., 58, 59
Kanitz, S., 162, 172–173
Katz, M., 17, 22
Keagy, D. M., 21
Keenan, R. G., 137
Kemeny, E., 21
Kemnitz, D. A., 5
Kenrick, G. W., 39
Kerka, W. F., 107
Klarman, H. E., 84
Kline, D. B., 41
Knowelden, J., 166, 168, 174–175
Kolb, L. H., 139
Kolesnichenko, T. S., 139
Korff, F., 39
Kotin, P., 120, 132, 136, 137, 138, 141
Krahl, V.E., 111
Kramer, G. D., 21, 22
Kreichelt, T. E., 5
Kuepel, R. E., 138
Kurland, L. T., 156
Kuschner, M., 140

La Belle, C. W., 135, 136
Lainhart, W. S., 137
La Mer, V. W., 118
Landahl, H. D., 114, 118
Landau, E., 159, 162, 164, 172–174
Landsberg, H., 38, 39, 40, 41
Langer, G., 71
Larrabee, C. P., 66, 67, 68, 69
Lawther, P. J., 153, 168, 169, 171, 175
Le Clerc, E., 69, 81
Lee, G., 113
Lee, R. E., Jr., 19, 26
Lehmann, C., 118
Lehmann, K., 117
Lemke, E. E., 12
Leonard, A. G., 154, 155
Linnell, R. H., 105
Lippmann, M., 17, 122
Lloyd, T. C., 167
Lodge, J. P., 19, 20, 23

Long, J. E., 135, 136
Ludwig, F. L., 19
Ludwig, J. H., 42, 43
Lunn, J. E., 166
Lynch, J. R., 137

Mac Phee, R. D., 26
Magill, P. L., 95
Mantel, N., 140
Manzhenko, E. G., 167, 174,–175
Marcus, S. C., 167
Mark, H. L., 106
Markul, I., 138
Markush, R. E., 157
Martin, A. E., 152, 153, 156, 171–172
Mateer, C. L., 40
McCaldin, R. O., 167
McCarroll, J., 150, 154, 156, 171–172
McConnell, W. J., 117
McCormick, R. A., 19, 37, 38, 42, 43
McCrone, W. C., 18
McCune, D. C., 93, 94
McDermott, M., 133
McKee, H. C., 105
McMullen, T. B., 16
McNerney, J., 119
Meeker, G. O., 11
Meetham, A. R., 40, 41
Megaw, W. J., 23
Meller, H. B., 69, 71
Metnieks, A. L., 23
Michelson, I., 81
Middleton, W. E. K., 51, 52
Mie, G., 10, 55
Miller, A., 135
Miller, E. C., 137
Miller, P. M., 94
Miller, W. S., 111
Milley, P. S., 123
Miner, M. L., 95
Mitchell, J. M., Jr., 43
Mitchell, R. I., 111
Moll, A. J., 136
Morrow, J., 105
Morrow, P. E., 111, 118, 119, 121
Mountain, I. M., 150, 156, 171–172
Mountain, J. D., 149, 156, 171–172
Mueller, P. K., 26, 57

Nadel, J. A., 17, 132
Nader, J. S., 19
Nagelschmidt, G., 69
Nau, C. A., 132
Neal, J., 132
Nelson, N., 113, 117, 118, 122
Neuberger, H., 40
Ney, F. G., 118
Noll, K. E., 55, 57
Nussbaum, R., 92

O'Connor, J. J., Jr., 81
O'Konski, C. T., 19
Orr, C., 23
Ortiz, H., 39
Ott, R. R., 105
Owens, J. S., 117
Ozolins, G., 12

Paccagnella, B., 167, 174–175
Pack, M. R., 93, 94
Pajenkamp, H., 92, 93
Palm, P. E., 119
Palmes, E. D., 113, 117
Parish, S. B. 89
Park, J. C., 21
Parker, A., 71
Pascua, M., 137
Pate, J. B., 19
Patterson, H. S., 117
Patterson, R. K., 19, 26
Pattle, R. E., 130, 131, 136
Paulus, H. J., 154
Pavanello, R., 167, 174–175
Payne, W. W., 138
Peirce, G. J., 89, 91
Pemberton, J., 157, 158, 159, 164, 172–174
Pesarin, F., 167, 174–175
Peterson, C. M., 54
Peterson, J. T., 43
Petrie, T. C., 72
Petrilli, F. L., 162, 172–173
Pfitzer, E. A., 123
Phillips, P. H., 95
Pickard, H. B., 19
Pilat, M. J., 54, 56
Pitts, J. N., Jr., 19
Plotkin, T., 113
Policard, A., 123
Pollack, L. W., 23
Potter, J. G., 41
Pregermain, S., 123
Preining, O., 11, 58, 59
Preston, R., St. J., 66, 69
Prindle, R. A., 164, 167, 173–174
Przemeck, E., 93
Pueschel, R. F., 10, 19, 54, 55, 56, 59
Purvance, W. T., 23
Pybus, F. C., 137
Pylev, L. N., 139

Quebedeaux, W. A., 105

Raymond, V., 92
Reed, J. I., 153, 154, 171
Rees, W. H., 71 ,72
Rehm, F. R., 24, 25
Reid, D. D., 162, 163, 165, 167, 172–173, 174–175
Reid, L., 135
Rich, S., 94
Rich, T. A., 23
Ridker, R. G., 84
Rinehart, W. E., 135
Robbins, R. C., 10
Robison, C. B., 101
Robinson, E., 19, 51, 52, 53, 58
Robinson, N., 38
Robson, C. D., 17
Roderick, W. R., 105
Roesler, J. R., 19
Ronald, G., 18
Rose, A. H., 24
Rossano, A. T., 105
Rowling, H., 26

Saffiotti, U., 139
Saito, Y., 117
Salem, H., 130, 132, 136
Samuels, S. W., 99, 101
Sanderson, H. P., 15, 22
Sanyal, B., 66, 69
Sauberer, F., 39
Sawicki, E., 138, 140
Sawyers, L. A., 113
Sayers, R. R., 117
Scheffer, F., 93
Schilling, F. J., 165, 173–174
Schnurer, L., 131
Schonbeck, H., 92
Schrader, J. H., 39
Schrenk, H. H., 134
Schueneman, J. J., 101
Schusky, J., 99
Scott, J. A., 152, 171
Scott, W. E., 105
Selikoff, I. J., 130
Seltser, R., 162, 163, 167, 172–173, 174–175
Sensenbaugh, J. D., 17
Seriff, N. S., 153, 171
Shabad, L. M., 139
Shaddick, C. W., 151, 171
Shaffer, N. R., 12
Shaver, J., 135
Sheesley, D., 11
Shelden, J. M., 17
Sheleikhovskii, G. V., 37, 38, 39
Sheppard, P. A., 37
Shubik, P., 139
Shupe, J. L., 95
Sievers, F. J., 94
Silverman, L., 23, 113
Sisson, L. B., 69, 71
Skidmore, J. W., 69
Slater, R. W., 17
Smith, B. M., 10
Smith, R., 16
Smith, W. S., 27, 101
Speizer, F. E., 168, 175
Spiegelman, J., 122
Sprague, H. A., 159, 161, 172–173
Spurny, K., 20
Stagg, J. M., 39, 40
Stalker, W. W., 20, 22, 101
Stanley, T., 138
Steiner, P. E., 138
Steinhauser, F., 39
Steinhubel, G., 91
Shembridge, V., 132
Stern, A. C., 19, 51, 52, 53, 58, 72
Stevenson, H. J. R., 19
Stewart, M. J., 139
Stocks, P., 157, 171–172
Stoeber, W., 17
Stokinger, H. E., 129, 130, 134
Stone, R. W., 162, 163, 167, 172–173, 174–175
Stout, A. P., 141
Stout, G. E., 42
Stratmann, H., 93
Strauss, W., 23
Strethlow, C., 122
Sullivan, J. L., 22
Sutton, O. G., 106
Swartztrauber, P., 19

Tabershaw, I. R., 137
Tabor, E. C., 71, 72, 139, 140
Taylor, J. R., 26
Tebbens, B. D., 72
Telford, J. W., 42
Thomas, B. G. H., 117
Thomas, M. D., 93, 94
Thompson, R. M., 117
Tice, E. A., 65, 71
Tinker, C. M., 168, 176
Tomashefski, J. F., 112
Tourangeau, F. J., 118
Tourin, B., 81
Toyama, T., 162, 167, 172–173, 174–175
Tracewell, T. N., 118
Transtrum, L. C., 93, 94
Tremer, H., 132, 136
Tufts, B. J., 23
Turk, A., 105, 106

Uhlig, H. H., 80
Underhill, D. W., 132, 136
Upham, J. B., 68

Valko, P., 43
Van Haut, H., 193
Van Wijk, A. M., 117
Venezia, R., 12
Verma, M. P., 165, 173–174
Vernon, W. H. J., 65
Verssen, J. A., 12
Vintinner, F. J., 132
Volz, F., 19, 37, 43
Von Zuilen, D., 150, 153, 158
Vorwald, A. J., 134

Wagman, J., 10, 19, 26
Wagner, W. D., 130
Walkenhorst, W., 117, 119
Waller, R. E., 71, 165, 169, 173–174
Walter, E. W., 150, 156, 171–172
Walther, J. E., 106
Walton, W. H., 17
Wang, C. S., 54, 58
Warren, W. V., 71, 72
Watanabe, H., 154, 167, 171, 174–175
Wedd, C. D., 131
Weibel, E. W., 114
Weinstein, L. H., 93, 94
Weisbrod, B. A., 84
Went, F. W., 53
Wentzel, K. F., 92
West, P. W., 19
Whitby, K. T., 23
Wicken, A. J., 162
Williams, J. D., 99
Williamson, J. B. P., 69
Wilms, W., 93
Wilson, I. B., 118
Winkelstein, W., 158, 172–173
Wiseley, D. V., 137
Wohlers, H. C., 106
Wolfson, P., 122
Wright, G. W., 167
Wynder, E. L., 19

Yancey, A. R., 113
Yant, W. P., 117
Yarmus, L., 113, 117, 118
Yocom, J. E., 69, 71

Zeidberg, L. D., 101, 156, 159, 161, 164, 172–173, 173–174
Zickmantel, R., 64, 173–174
Zwi, S., 132

SUBJECT INDEX

A

Acrolein toxicity, 135, 136
Adhesion properties, 8–9
Adhesive dustfall collectors, 17
Adsorbed substances
  discussion of, 130
Adsorption
  role in odors, 107
  role in toxicity, 130
Aerodynamic factors
  respiratory tract, 113–114, 115–116
Aerosols
  pulmonary effects, 131–137
  reduction of toxicity, 130–131
  toxicity, 129
Age
  relationship to mortality, 148, 151, 158, 160
Air pollution episodes, 15, 150–154
Alveolar deposition, 119
Ammonia toxicity, 122, 135
Animal odors, 107
Aromatic hydrocarbons as carcinogens, 137–141
Asbestos
  toxicity of, 129–130, 150
Automobile exhaust emissions, 26
Automobiles
  corrosion of, 71

B

Beryllium
  toxicity of, 129–130, 150
Birmingham, Alabama
  public opinion survey, 101
Bronchitis
  morbidity studies of, 156, 161–169
  mortality studies of, 150–154, 156–161
Brownian motion, 9, 113
Buffalo, New York
  mortality studies, 158, 160, 161, 170, 171
  public opinion survey, 101
Building materials
  deterioration of, 69
Buildings
  deterioration, 73–74

C

Canada
  morbidity studies, 163, 164, 173
Cancer
  lung, 137, 141, 157–162
  mortality, 157, 160–162
  stomach, 158, 161–162
Carbon black
  toxicity of, 132
Carbon particles
  toxicity, 135
Carcinogenic hydrocarbons, 137–142
Carcinogens
  discussion of, 137–142
Cascade impactors, 17, 19
Cement plants
  emissions from, 24, 25–26
Cement-kiln dust, 89–93
Channel black effects resp. tract, 132
Chemicals
  odors from, 107
Chicago mortality studies, 150
Children
  morbidity studies, 165–167
Chlorides
  corrosive effects, 71
Clearance model
  respiratory system, 120–121
Clearance of particulate matter in the respiratory system, 121–123
Climate
  effects of particulate on, 35, 42–44
Climatic change
  world-wide, 42–44
Coal combustion
  effects of smoke on animals, 131–132
  emission from, 24, 27
Coal combustion gases
  corrosive effects, 69–70
Coal dust
  deposition, 117
  physiological effects of, 133
Combustible waste odors, 107
Combustion odors, 107
Combustion products
  corrosive effects, 69–70
Computer techniques of sampling, 19
Copper alloys
  corrosion of, 68–69
Corn oil particles
  deposition, 118
Corrosion of metals, 65–69, 72–74
Costs of air pollution damage, 79–84
Cotton
  soiling and deterioration of, 72, 74
Crust formation on plants, 89–93
Cyclonic collectors, 17

D

Deposition
  respiratory system, 112–119
Deposition (dust) on vegetation, 89–96
Desorption
  odorants, 106–108
Deterioration of materials, 65–74
Detroit
  mortality studies, 150, 154, 171
Diurnal variation
  particulate, 40, 41
Dublin, Ireland
  mortality study, 154, 155
Dust
  corrosive effects, 65–74
  effects on resp. tract, 112–119
  effect on textiles, 71–72, 74
Dust components, 92–93
Dust mineral
  deposition, 117
Dustfall
  description of, 11, 16–17
  seasonal variation, 39
  typical urban values, 17
Dustfall collectors, 17–19
Dustfall jars, 17

E

Economic effects, 79–85
Economical level
  relationship to mortality, 156–158, 159–162, 163, 164, 167, 170, 176
Electrical instruments
  corrosion of, 69
Electron microscopy, 20, 23
Elutriators, 17
Emission factors, 24
Emission inventories, 12

England
  morbidity studies, 154–156, 161, 162, 163, 164–169, 171–176
  mortality studies, 150–153, 154, 156–159, 171–176
Epidemiological studies, 150–154
Exacerbation of chronic diseases, 148–149, 154–156, 161–176
Extinction
  dependence on particle size, 55

F

Ferric sulfate
  toxicity, 132–133
Filters
  sampling, 20–22
Fluorides
  effects on vegetation, 93–94
Fluorosis
  animal, 95
Fog
  correlation with mortality, 150–153
Food odors, 107
Formaldehyde
  toxicity, 122, 135, 136
Foundry
  odors from, 107
Foundry dust
  effects on vegetation, 94
France
  economic effects of air pollution in, 81
Fuel oil combustion
  emissions from, 24

G

Gas and particulate mixtures
  synergistic pulmonary effects, 134–137
Gases
  toxicity of, 134–137
Great Britain
  economic effects of air pollution in, 81
  medical effects of air pollution, 150–159, 161–169, 171–176

H

Haze
  particulate size distribution of, 52–53
High-volume sampler, 22–23
Hydration process
  dust crust, 91–92
Hydrocarbons as carcinogens, 138–141
Hygroscopic particles
  effect on pulmonary irritants, 136

I

Illumination
  seasonal variation, 37
Industrial odors, 107
International Standard Calibration Curve for reflectometers, 21
Irkutsk (U.S.S.R.)
  morbidity studies, 167, 174
Iron
  corrosion of, 65–69
Iron dust
  effect on textiles, 72
Iron oxide
  effects on vegetation, 94
Italy
  morbidity studies, 162, 167, 173, 174

J

Japan
  morbidity study, 162, 167, 173, 174–175
  mortality study, 154, 171

L

Leaves
  dust effects on, 89–96
Light absorption, 52–53
Light scattering, 9–10, 37–38, 52–53, 54–58
Linen
  soiling and deterioration of, 71–74
London
  morbidity studies, 154–156, 162, 163, 165–167, 168–169, 172, 173, 174, 175
  mortality studies, 56–158, 171–172
Lung deposition, 118–119
Lung dynamics
  model of, 114–116

M

Magnesium oxide
  effects on vegetation, 94
Membrane filters, 20
Messthelioma, 129–130
Metals (see specific metal)
Meuse Valley
  mortality studies, 150
Mie solutions, 54–55
Morbidity
  correlation with pollution, 148–149, 154–156, 161–176
  in children, 165–167
  incapacity for work, 164–165
Mortality
  correlation with acute air pollution episodes, 148, 150–154
  exposures to air pollution, 148, 150–154, 156–161, 169–176
  Long term, 156–161
Motor vehicles
  emissions from, 26
Municipal incineration
  emissions from, 24–25

N

Nasal fractionation, 118
Nashville, Tennessee
  morbidity studies, 164, 173
  mortality studies, 160, 161, 173
  public opinion survey, 100–101
Nephelometer, 19
New Hampshire (Berlin)
  Morbidity study, 164, 173
New York City
  morbidity studies, 154–156, 165, 170, 171, 173, 176
  mortality studies, 156, 170–171
Nickel
  corrosion of, 67–69
Nitrogen dioxide
  toxicity, 135
Nucleation properties, 8–9
Nuisance surveys, 99–102

O

Odors
  association with particles, 106–107
  emission sources, 107
Open hearth furnaces
  emissions from, 24
Optical density, 20–22
Optical properties, 9–10
Osaka
  mortality study, 154, 171

P

Paint odors, 107
Painted surfaces
  deterioration of, 71, 72–74
Particle formation mechanisms, 10–11
Particle-gas reactions, 10
Particle size
  role in measuring techniques, 149
  role in pulmonary deposition, 114, 118–119
  role in pulmonary effects, 132–133
  distribution, 58, 117
Pennsylvania communities
  morbidity study, 167–168, 175
Philadelphia, Pennsylvania
  economic effects of air pollution in, 83

216

Photochemical reaction model, 10
Photometry, 20
Photomicrographic atlas, 19
Physiological response, 132–134
Pittsburgh, Pennsylvania
  economic survey of pollution damage, 180
Plants
  dust effects on, 89–93
Pneumocoiosis, 122, 131
Pneumonia
  mortality, 157
Polynuclear aromatic hydrocarbons
  carcinogenicity of, 137
Post office employees
  morbidity studies, 163
Precipitation
  influence of particulates on, 40–42
Public opinion surveys, 99–102
Pulmonary flow resistance, 132–134
Pulmonary function
  alterations in, 132–134

R

Race
  relationship to mortality, 156
Rainfall (See Precipitation)
Refinery
  odors from, 107
Reflectance, 21
Reflectometer, 21
Respiratory illness
  relation to smoke level, 148, 162–167
Respiratory system
  models of, 114–116
Respiratory tract
  anatomy of, 111–112
Retention
  human vs. animal respiratory system, 119
  respiratory system, 112–119
Rotterdam
  morbidity study, 168, 175
Rusting, 65–69, 72–74

S

Salford, England
  morbidity studies, 164
  mortality studies, 158–159
Sampling methods, 17–23
Scattering coefficient, 55–58
Settling velocities, 16
Sewage
  odors from, 107
Sex
  relationship to mortality, 156
Sheffield, England
  morbidity studies, 167, 168, 170, 174
Smoke
  corrosive effects, 72–74
  toxicity of, 150–154
Smoke plumes
  optical properties of, 53
Smoke shade
  correlation with mortality, 154
Smoking
  cigarette, 137, 141, 149–150, 158, 161, 162, 163, 166
Sodium chloride
  corrosive effects, 71
Solar radiation
  effects of particulates on, 35–38
  physical factors effecting, 38–39
  seasonal variations, 39–40
  weekly variations, 39
Soot
  corrosive effects, 65–74
  effects on textiles, 71–74
  effects on vegetation, 89–94
Sorption properties, 8
St. Louis, Missouri
  economic effects of pollution in, 83
  public opinion survey, 99–100
Steel
  corrosion of, 65–69, 72–74
Stokes law, 5–6
Sulfur dioxide
  corrosive effects, 65
  odor, 107
  toxicity of, 130–131, 132, 136–137
Sulfuric acid
  effects on vegetation, 94
Sulfuric acid manufacturing
  emissions from, 24
Supersaturation
  nuclei, 23
Surface coatings
  deterioration of, 69–71, 72–74
Surface properties, 8–9
Surveys of damage, 79–84
Suspended particulate
  means for urban areas, 11, 13–16
Synergistic effects
  gases and particulate, 135
Synthetic textiles
  soiling and deterioration of, 71–74
Syracuse, New York
  economic effects of air pollution in, 83

T

Tape samplers, 20–22
Telephone workmen
  morbidity studies, 162, 163
Tetrahydro naphthalene
  toxicity, 131
Textiles
  soiling and deterioration of, 71–74
Toxicity
  adsorbed substances, 130
  intrinsic of particulates, 129–130
  reduction of, 130–131
Transmissivity
  variation with climate, 36
Transmittance, 20–21
Trees
  dust effects on, 90–92
Triphenyl phosphate
  deposition, 117
Turbidimetry, 19
Turbidity, 37

U

Upper Ohio River Valley
  economic survey of pollution damage, 81, 82

V

Varnished surfaces
  deterioration of, 69–71, 72–74
Vegetables
  dust effects on, 89–96
Visibility, 51–61
  effect of fog upon, 53–54
  effects of natural aerosols on, 53
  effects of particulates on, 36–38, 53–53
Volatile particles, 106

W

Washington, D.C. (Suburban)
  economic effects of air pollution, 83
Waste decomposition
  odors from, 107
Wool
  soiling and deterioration of, 71–74

X

X-ray diffraction, 19

Z

Zinc
  corrosion of, 66, 67–68, 72–74
Zinc dust
  effect on textiles, 72
Zinc salts
  corrosive effects, 71

ACKNOWLEDGEMENTS

The following sources, in most instances the copyright holders, have granted permission to the National Air Pollution Control Administration to include the following figures and tables in the Air Quality Criteria:

Table 1–1.—Los Angeles data courtesy of Los Angeles County Air Pollution Control District

Table p. 26.—Chemical Processing Engineering, London

Table p. 27.—American Society of Mechanical Engineers, New York, N. Y.

Table 2–2.—The Gray Printing Company, DuBois, Pennsylvania

Tables 3–1, 8–1.—Air Pollution Control Association, Pittsburgh, Pa.

Table 3–2.—Reinhold Book Corporation, New York, N. Y.

Table 4–1.—Iron and Steel Institute, London

Tables 4–2, 4–3.—American Society for Testing & Materials, Philadelphia, Pa.

Table 5–1.—Mellon Institute, Pittsburgh, Pa.

Table 10–2.—American Industrial Hygiene Association, Detroit, Mich.

Tables 10–3, 10–4, 11–2, 11–3, 11–4.—American Medical Association, Chicago, Ill.

Table 10–5.—American Association for the Advancement of Science, Washington, D.C.

Table 10–6.—National Tuberculosis & Respiratory Disease Association, New York, N. Y.

Table 11–1.—Pergamon Press, Inc., New York, N. Y.

Figure 1–1.—Academic Press, Inc., New York, N. Y.

Figures 1–3, 2–2, 3–1, 3–4, 3–5, 3–6, 9–4, 9–5, 9–6, 9–7.—Pergamon Press Inc., New York, N. Y.

Figure 1–4.—American Industrial Hygiene Association, Detroit, Mich.

Figures 2–1, 3–7, 7–2.—Air Pollution Control Association, Pittsburgh, Pa.

Figures 2–3, 2–4, 11–2.—Controller, Her Majesty's Stationary Office, London, England

Figures 2–5, 3–1, 3–3.—American Meteorological Society, Boston, Mass.

Figure 3–8.—American Chemical Society, Washington, D. C.

Figure 4–1.—Iron and Steel Institute, London, England

Figure 4–2.—American Society for Testing & Materials, Philadelphia, Pa.

Figure 4–3.—District of Columbia Health Department, Washington, D. C.

Figure 5–1.—Environmental Health & Safety Research Associates, New Rochelle, New York

Figures 6–1, 6–2, 6–3.—Ellis F. Darley, University of California, Riverside, Calif.

Figure 11–1.—Royal Society of Health, London, England

Figure 11–3.—Royal Dublin Society, Dublin, Ireland

Figure 11–7.—American Medical Association, Chicago, Ill.

Figure 11–8.—British Medical Journal, London, England

☆ U. S. GOVERNMENT PRINTING OFFICE : 1969—347-411/32